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THE MEDICAL CLINICS
OF
NORTH AMERICA

VOLUME 2 NUMBER 5

CLINIC OF DR. HENRY A. CHRISTIAN

PETER BENT BRIGHAM HOSPITAL

CUTANEOUS PIGMENTATION, JAUNDICE, PALPABLE LIVER AND SPLEEN, AND ASCITES

December 9, 1918.

DR. CHRISTIAN: The patient (P. B. B. H., Med. No. 9841) that I wish to show this morning is a woman forty-four years of age, born in Ireland and having lived in Ireland until six years ago, when she came to Boston. She has lived in Boston since then. (To student who had been told to examine patient during demonstration of *x*-ray plates from another patient): Mr. ——, tell us in brief what you make out in this patient.

STUDENT: She has a rather dark skin, which she says is not particularly darker at present, she having always been rather dark, but perhaps not as dark as she is now. Her eyes are yellow, which is not due to pigmentation, but there seems to be fat over the scleræ.

DR. CHRISTIAN: Yes, there is a slight layer of fat which is yellowish, but, in addition, there is a slight general yellow color to the scleræ.

STUDENT: The mucous membrane seems normal. The hands are unpigmented. The chest and abdomen are a deep brown, with scars.

DR. CHRISTIAN: What did you say about her hands?

STUDENT: They are pretty white as contrasted with the face.

DR. CHRISTIAN: The face is definitely darker than the hands, but yet you can see, as contrasted to my hands and the hands

of the nurse, that there is also pigmentation of the hands, rather more about the forearms and elbows than in the hands, and the palms of the hands also show a slight degree of pigmentation. Pigmentation is more marked on the face than it is on the hands. It is more marked on the forearm than it is on the hand, and possibly it is increased a little bit in the upper arm as contrasted to the forearm.

You said the pigmentation was more marked on the abdomen than it was on the hand. What does the fact that the hands are less pigmented than the trunk suggest?

STUDENT: That it is not any form of pigmentation which depends on the action of sunlight for a deposition of the pigment.

DR. CHRISTIAN: Yes, there is some factor other than exposure to sunlight. If it were more marked on the face and hands, you would suspect exposure to the sun, but being less marked on the hands and distinctly more marked on the protected parts as well as on the face, which is unprotected, pretty well rules out pigmentation from exposure to sunlight. In the same way, it is always well in a case of pigmentation to see whether there is a sharp line of demarcation between the forehead and the adjacent scalp which is protected by the hair. In many cases there is often a definite difference in pigmentation at the hair-line between people exposed to the sun and those not so exposed, and particularly where they have worn hats do you get the change. Her scalp is pigmented, so she has this general pigmentation which, on the whole, is more marked on the face, neck, and thorax than it is on the hands. (Inspection of patient's feet.) There is not as marked pigmentation on the feet as on the face, but it is probably a little bit more marked on the feet than on the hands. While we are looking at her feet observe this distinct subcutaneous edema of the feet and of the lower leg.

What else did you make out in the brief time you had to examine her?

STUDENT: The abdomen is distended, apparently with fluid, because there is a fluid wave and dulness in both flanks and extending over the whole abdomen with the exception of a small area at the top, which is tympanitic.

DR. CHRISTIAN: The abdomen is distended, the greater part of the abdomen is dull, tympany is limited to a small area in the center, there is a fluid wave, and that is pretty good evidence of fluid. Did you feel the liver or spleen?

STUDENT: I could not get in; the abdomen is too tense.

DR. CHRISTIAN: The abdomen is tense, so that the liver and spleen were not made out. We cannot feel the liver and spleen when her abdomen is tense this way. She has been tapped, and after tapping the liver edge at times has been felt, not markedly enlarged, and the spleen has been felt, apparently the spleen, in a proportionate sense, being a little bit larger than the liver, but neither one is very large. The abdomen has not a uniform pigmentation, but there are pale areas where there is vitiligo or absent pigmentation, so she has this brownish pigmentation with areas showing absent or decreased pigmentation. There is a pale area on the abdomen about the size of a 5-cent piece, and there are smaller areas of that type scattered over the trunk, so we have a generalized pigmentation, brownish in color, more marked on the trunk than on the forearms or lower legs, present throughout the skin to a certain degree except in scattered small irregular areas, where there is an abnormal absence of pigmentation; we have a combination of cutaneous pigmentation and small areas of absent pigmentation or, as we call it, vitiligo. In addition, she has edema of the feet and ascites, and in the conjunctivæ a small trace of jaundice, not very marked; she has no evidence of jaundice as far as the skin goes.

Did you get a chance to examine her thorax?

STUDENT: I went over it hurriedly. The heart sounds below the nipple are very distant, and in the region of the second interspace there is a quite marked systolic murmur which is best heard there, but also is heard on the right side. The chest seemed normal on auscultation and on percussion in front. Behind, the sounds seem somewhat diminished on the left side.

DR. CHRISTIAN: Is that all over the lung or toward the base?

STUDENT: At the base, and, with the questionable dulness, the tactile fremitus seemed diminished at the same point.

DR. CHRISTIAN: That is quite characteristic. There is a

systolic murmur, but the heart is not enlarged. With that amount of fluid in the abdomen the heart naturally is encroached upon by the high diaphragm and somewhat disturbed in relation to the chest wall, so you do not get the maximum sounds where you might expect them. In the same way there are decreased breath sounds and dulness at the base behind, apparently a little more on one side; as far as we can judge, that is simply a high diaphragm crowded up against the heart in front, and, in the same way, on the lungs behind, giving a higher line of dulness than you ordinarily get, but the heart and lungs are essentially normal.

Since she has been in the hospital she has had a somewhat irregular fever, going at times fairly high. It is practically never normal throughout the twenty-four hours. Ordinarily it runs from a little below normal to about 100° F., with occasional higher elevations. When she first came in she ran a high temperature, at one time going up to 104° F., with a pulse a little fast—between 110 and 115. Recently her pulse has been running between 80 and 90. The respiration has been pretty uniformly between 20 and 25 for fourteen to fifteen days, and then it has run about 20 since, so from the chart there is no evidence of any disturbance in the cardiac or respiratory mechanism, but there is a slight irregular fever.

I will give you a little more about her history. There is no alcoholic history. She has been a person who has used a rather large amount of tea—six cups of tea daily. She has worked as an attendant nurse. Previous to five years ago she worked very hard, with long hours, but stood her work well. For many years she has been living with an elderly lady where the work has been very easy and congenial, she having her afternoons and evenings to herself. She had been doing this work up to the time of her admission. As I have already said, she was born in Ireland and lived there until six years ago.

There is no history of previous illness. As she expressed it, she was an unusually healthy woman. While living in Ireland she very frequently was closely associated with advanced cases of pulmonary tuberculosis, but gives no history of any symptoms

herself suggesting tuberculosis. There has been no disturbance in her head, eyes, or ears. In regard to her nose, she has had no discharge or any obstruction, but has had attacks of epistaxis during the past eight years associated, as she thinks, with her menstruation. Not every month, but almost six months out of the year she has a sudden nosebleed, mostly from the left nostril, just before the onset of her monthly sickness. She never loses more than 1 or 2 tablespoonfuls of blood at one time. These attacks of epistaxis ceased entirely two years ago until four months ago, when she had a single attack with her last menstrual period. She has had false teeth for two years. I do not know about her teeth prior to that. There is no cardiorespiratory disturbance in her past history.

The patient has always had a very good appetite and has eaten three heavy meals daily. Her bowels have always been regular without cathartics, averaging one or two movements daily. She has never felt nauseated or had gaseous eructations, although she has been troubled by gas per rectum at times, but this has not been present in the past four months. She never has had vomiting, hematemesis, distress, or colicky pains. Her color has always been dark, and frequently, since the age of twenty-eight, she says her skin has been dark and yellowish at intervals. So far as she knows her scleræ have never been yellow prior to the present illness. There is no history of diarrhea or clay-colored stools previous to the present illness. She gives a history of moderate smarting discomfort at stool suggesting the possibility of hemorrhoids. Prior to the present illness she had to get up from two to three times at night to pass her urine. During the past four or five months she has had no nocturia and thinks she has been passing smaller quantities of urine at a time. (Her chart confirms that since she has been in the hospital.) There has been no other abnormality noted in regard to her urine.

There is no history of any venereal disease and nothing to suggest a previous gonorrhea or syphilis. Previous to coming to the place at which she works at present her sleep was very irregular, but for the past three years she has slept very well.

There has been nothing abnormal about her history of sleep until quite recently, and I will bring that out in connection with the present illness. I presume the irregularity of sleep before that refers to her work as an attendant nurse, and that she had to get up from time to time in the course of her duties and so did not have regular sleep. The nurses abroad are rather apt to be on the job for twenty-four hours, and in taking care of a patient sleep in the room with the patient at night. They do not follow the American system of having a day and night nurse.

Her catamenia commenced at the age of fourteen and has always occurred with great regularity until four months ago, when she had her last period. She has always flowed abundantly, it lasting seven days. She has never suffered pain or had any symptoms during or after her periods. There was nothing abnormal about the catamenia except the associated nosebleeds already described. Her best weight was 141 pounds a month ago. Her average for the past ten years has been 140 pounds. She says she has been growing thin for at least two months, and all her friends remarked her loss of flesh, and yet she weighs as much as she ever did, weighing about 141 pounds on admission. Of course that is due to the fact that the accumulating ascites and simultaneous loss of fat and muscle substance have apparently gone parallel, so that now she weighs her best normal weight with what she has noted—loss of flesh—and the loss of flesh made up by a gain in the ascitic fluid which we have noted.

As to her present illness—six months ago the patient was perfectly well as far as any symptoms were concerned. She has had no pain, no subjective signs or discomfort except itching of the skin and increased discomfort from an enlarged abdomen and swelling of the legs, to be described later. These symptoms began six months ago, and prior to that she had no symptoms, and those are the only symptoms she has had—itching of the skin, enlargement of the abdomen, and swelling of the legs. At present she feels well, and only yesterday stood on the street all day long viewing the parade (that was the peace celebration parade). The first trouble the patient noticed was itching and irritation of the skin which was probably most severe five to six

months ago. So intense was the itching that she excoriated herself from scratching the lesions. On the skin of the abdomen there was a sensation as though small insects were burrowing into the flesh. Besides on her abdomen the irritation was intense on the arms and legs. She thinks she was somewhat jaundiced at this time. She consulted a doctor, who told her that she had liver trouble, but gave her little advice as to the nature of the condition. The itching on the skin has diminished gradually and has given very little discomfort in the past month. At present there is very slight irritation. About six months ago the patient, though feeling well, noticed that she could sleep for three to four hours in the afternoon, so that she regularly did this. She was able to keep awake if she had any work that she was obliged to do. Her skin was of a rather yellowish hue, but this was not pronounced.

One month ago, while taking care of a patient sick with influenza, where she worked very hard, she suddenly became aware of a considerable swelling of the ankles and legs and her knees were particularly swollen. The swelling was hard and did not yield to pressure. At this time she consulted a physician, who told her that he was unable to account for it. The abdomen was swollen at this time and she says that during the following week she noticed it becoming rapidly distended. She doubts if it was a gradual increase in size which she had failed to notice, but regards her distention as having come on rapidly in the course of a week's time. According to her statement her jaundice increased in the past month, but she did not notice the scleræ to be yellow until entrance to the hospital, when her attention was called to it. On one occasion one month ago she noticed her urine to be very dark. She has always observed her urine, and says that it has been dark at intervals since then. About the same time her stool was of a light yellow color and she noticed a similar condition only once since, and that was the morning of admission. The patient says that she has never had any pain at any time and that she feels strong and healthy. She believes her flesh has fallen away considerably in the last few months, and she thinks that the only reason her weight is

maintained is because of the swelling of her abdomen. Having been thrown with patients as an attendant, though not a graduate nurse, that history probably represents a more intelligent observation of herself than we generally get in our patients, and it is a pretty clear history.

The physical examination is essentially negative except for the points that have been brought out, that is, pigmentation and fluid in her abdomen—she has been tapped on three occasions. On November 13th 3900 c.c. of yellow turbid fluid was removed, having a specific gravity of 1005, 5.8 grams of albumin per liter, quantitated with the Esbach method, which is only moderately accurate, no bile, no urobilin, 280 cells per centimeter; lymphocytes, 45 per cent.; mesothelial cells, 55 per cent. She was tapped again two days later and 1800 c.c. of the same kind of fluid was removed, specific gravity 1008, cell count 2670, 89 per cent. polymorphonuclears, 11 per cent. lymphocytes. The albumin in this specimen was slightly less, 4.8 grams per liter. On December 2d 3500 c.c. were removed, giving a specific gravity of 1006, 8 grams of albumin per liter, and 200 cells, 50 per cent. of which were lymphocytes, 47 per cent. mononuclears, in other words, mesothelial cells, and 3 per cent. polymorphonuclears. In the counting chamber it was observed that some of the cells contained rather numerous brown pigment granules.

She has not any leukocytosis. There is a slight degree of anemia; 4,008,000 red cells, with a hemoglobin of 85 per cent. The differential count is normal and there is nothing abnormal about the red cells. Her urine specific gravity has averaged from 1010 to 1030 at different observations. Occasionally it contains the slightest possible trace of albumin, but is usually free from albumin. It usually contains a very small amount of glucose. From time to time it has given a positive bile test. There are no red cells in the urine in most of the examinations, sometimes an occasional one. A few white cells are ordinarily found, but no casts have been found in a number of observations. One stool examined on November 14th was small, dark, fluid, no gross blood, pus, or mucus. Benzidin reaction was negative. Nothing abnormal was found microscopically in the

stool. On November 25th a stool was examined, which was clay colored, soft, unformed, no gross blood, pus or mucus, negative benzidin reaction. On the 26th the stool was clay colored, soft, unformed, no gross blood, pus or mucus, benzidin negative. There was a certain amount of diarrhea—four stools that day. Since then a number of stools have shown a negative benzidin test. Fairly abundant fatty acid crystals were noted in a number of the stools. The Wassermann reaction was negative. The blood-pressure is 140 systolic and 90 diastolic.

Mr. ——, what should you say about the diagnosis? What would you consider as the probable condition with that history and that physical examination?

STUDENT: Jaundice of one type or another.

DR. CHRISTIAN: Yes, the patient has jaundice. Of what type do you think it is?

STUDENT: The chronic type or probably a sort of catarrhal jaundice.

DR. CHRISTIAN: Do not say "catarrhal" unless you know what it means, and that you really mean it at this time. What do you mean by "catarrhal jaundice"?

STUDENT: Jaundice due to catarrhal inflammation of the bile-ducts, and possibly extending into the liver along the smaller ducts.

DR. CHRISTIAN: Catarrhal jaundice is a pretty definite clinical entity—jaundice without any evidence of any gross lesion in the liver, coming on acutely, usually associated with or preceded by a gastro-intestinal upset, moderate in nature, accompanied by fever in the early stages and the fever and gastro-intestinal upset clearing up, but the jaundice persisting over relatively a short time, a few days to a few weeks. Here is a patient that has had jaundice for a considerable length of time, so it is not catarrhal jaundice, and you do not want to use that term. What evidence have you as to a cause of the jaundice? What evidence have you in the patient of any other abnormality besides jaundice?

STUDENT: There is the cardiac lesion.

DR. CHRISTIAN: Which you said she did not have. Why do you say she has a cardiac lesion?

STUDENT: She has a systolic murmur, heard at the base.

DR. CHRISTIAN: What is the presumptive evidence about systolic murmurs at the base?

STUDENT: That they are functional.

DR. CHRISTIAN: Yes; so there is not much to stand on as evidence of a heart lesion. She might have a heart lesion, but with those findings as regards the murmur the burden of proof is on the lesion side. The presumptive evidence is that any patient with a basal systolic murmur has a normal heart until you prove that it is diseased in some way, decreased in function or increased in size. What does the accumulation of fluid in the abdomen suggest?

STUDENT: A sclerotic liver, syphilitic or alcoholic.

DR. CHRISTIAN: What term do we generally use when the liver is sclerosed?

STUDENT: Cirrhosis.

DR. CHRISTIAN: Yellow—it is an old term that goes back a great many years. It was used for the color of the liver, but, as the cirrhotic liver is not always yellow, when we use the term "cirrhotic," we mean now a liver with increased connective tissues in it and not the original meaning of the term. Why do you say that she probably has cirrhosis?

STUDENT: It is suggested by the ascites and interference with the portal circulation. The edema of the legs would not be so suggestive of it.

DR. CHRISTIAN: How would you explain the edema of the legs here?

STUDENT: Possibly the fluid in the abdomen would interfere with the return from the legs by pressure on the femoral veins.

DR. CHRISTIAN: Are the femoral veins in the abdomen?

STUDENT: As they pass up they are.

DR. CHRISTIAN: After they pass up, what are they?

STUDENT: The inferior vena cava.

DR. CHRISTIAN: I mean as they pass up to enter the inferior vena cava. There is the inferior vena cava (illustration on the board) and here are the femorals, what is in between?

STUDENT: You have the femorals and then the iliacs. There

are two sets, the common dividing into the external iliac and the internal iliac.

DR. CHRISTIAN: You have the inferior vena cava, the common iliac and the external and internal iliacs, and pressure on those in the abdomen will cause edema of the legs. What is it that causes stoppage of the circulation to the legs—what is the mechanism?

STUDENT: Distention of the abdomen probably is sufficient; that is, stretching of the abdominal wall is sufficient, though the pressure probably does not reach that point.

DR. CHRISTIAN: What point would it have to reach?

STUDENT: Complete closure of the veins, which would necessitate the overcoming of the various factors that produce venous circulation.

DR. CHRISTIAN: What is the pressure in the inferior vena cava?

STUDENT: Negative pressure.

DR. CHRISTIAN: What keeps the circulation from stopping if the pressure in the vein is low? Of course in the lower part of the inferior vena cava and iliacs the pressure is not a negative pressure, but is a low pressure and is a slightly positive pressure. That being so, why are not those veins blotted out and the circulation stopped?

STUDENT: The action of the valves and the pumping action of the diaphragm prevents it, and there is slight pressure from the arterial circulation.

DR. CHRISTIAN: Yes, slight pressure from all of those things come in. First, there is not any interference with the onward flow of the arterial supply. The arteries carry blood to the legs which must come back and can go against very considerable resistance as long as there is a good driving impulse in the heart forcing the arterial circulation. Why are the arteries more resistant than the veins?

STUDENT: They are deeper in.

DR. CHRISTIAN: What other difference?

STUDENT: They are thicker walled and have higher internal pressure.

DR. CHRISTIAN: They are much thicker walled and so rather better protected. If the arteries and veins were built alike and had the same pressure these circulatory disturbances from accumulating abdominal fluid would be a much more serious matter. As a rule you get what this patient has, simply a moderate degree of edema of the legs except that sometimes, with interference to the circulation and slowed venous circulation, there is a tendency to thrombus formation, and you get thrombi forming either in the veins in the abdomen or down the leg, and you get added to that picture venous thrombosis. These patients not infrequently have thrombosed veins in addition to having edema. The edema of the legs is most probably due to the ascites. How could you prove whether it was due to the ascites or not?

STUDENT: It ought to be reduced, if due to the ascites, when the abdomen was tapped.

DR. CHRISTIAN: If you mechanically can remove the fluid and keep it from recurring, in a short time the edema of the legs will disappear. Practically it is frequently rather difficult to carry out that test because the abdomen fills so promptly after it is tapped that it fills up again before the edema has time to drain away from the legs, but when there is a question of edema of the legs being due to ascites we observe to see whether tapping of the abdomen relieves the edema.

Did that happen in this case, Dr. ——?

DR. ——: Yes, it did.

DR. CHRISTIAN: It has been relieved after tapping, and the edema gets more marked when the abdomen becomes more distended. We have evidence of a certain degree of jaundice which suggests interference with the biliary function and points to some hepatic lesion. We have the ascites, which suggests that there is interference with the portal drainage, and that suggests either a lesion in the liver or a lesion in the portal vein that is obstructive in nature like thrombosis of the portal vein or disturbance in the circulation through the liver, such as is produced by cirrhosis, so the jaundice and ascites point to cirrhosis of the liver. The liver is a little enlarged, which is in

accord with the idea of cirrhosis, but does not prove cirrhosis. The spleen is moderately enlarged. What explanation could you give of that? In other words, what causes enlargement of the spleen?

STUDENT: In this case it seems as if with the liver cirrhotic the back-pressure might cause a certain amount of congestion of the spleen and enlargement.

DR. CHRISTIAN: Yes, the liver may be cirrhotic and the spleen simply enlarge on account of the congestion—passive congestion—brought about from interference in the portal circulation. What other changes cause enlargement of the spleen besides back-pressure from a cirrhotic liver?

STUDENT: Various conditions of hyperplasia from chronic or acute infections.

DR. CHRISTIAN: Yes, various chronic hyperplasias. Marked increase in the size of the spleen is the result of chronic hyperplasia and that effects both the connective tissues in the spleen and the amount of cellular elements in the spleen. Very often with cirrhosis of the liver the spleen is enlarged beyond what you would expect from portal obstruction, and we say that, because the same kind of obstruction, brought about from poor circulation on account of a heart condition, having just the same effect as far as back-pressure on the portal system is concerned, as a rule, does not produce any marked enlargement of the spleen, but produces a moderately enlarged spleen, whereas cirrhosis of the liver patients may have a markedly enlarged spleen. You have already seen a case of this type with enlarged spleen. Dr. Wolbach showed you one that belonged in the group of Banti's disease in which there was some other cause for the enlargement of the spleen than enlargement simply from back-pressure. In this patient the splenic enlargement may be only the result of cirrhosis of the liver or may be from some chronic inflammatory change leading to hyperplasia. Is there anything in this history to suggest in this particular case that the change in the spleen is one or the other?

STUDENT: The blood is essentially negative, which would tend to exclude infections, as there is no leukocytosis.

DR. CHRISTIAN: Chronic infections probably would not give a leukocytosis, and chronic splenic hyperplasia usually has a normal blood-picture. What is there in the patient's history? How long has the abdomen been enlarged? Her abdomen swelled three weeks ago and her legs were noticed to be swollen four weeks ago. She thinks these changes came on pretty definitely at that time. Assuming that the edema of the legs is due to change in her abdomen, and the abdomen showed some swelling when the legs were noticed to be swollen, that would take it back a month, or, to make it prior to that a reasonable length of time, we say the abdomen has been enlarged for six weeks; that is hardly long enough, from the point of view of passive congestion, to cause a moderately enlarged firm spleen, so if her spleen is definitely enlarged and firm, as suggested by some of the observations, presumably there has been some hyperplastic process going on in the spleen and it is not simply a question of back-pressure.

What about the color of the skin apart from jaundice? You have evidence of jaundice with a little yellow color to her scleræ. We have found bile present in her urine, and that is further borne out by the fact that there is a decreased amount of bile in her stool. Some of the stools are clay colored without being essentially fatty stools. Bile, however, is not absent from the stool. When tested for it is found. At times it is present in enough quantity to color the specimen. How about the pigment in the skin?

STUDENT: It is darker than the ordinary pigment of jaundice. It suggests more Addison's disease. It is brownish almost.

DR. CHRISTIAN: It is a dark pigment, not particularly a yellow pigment, such as we see in jaundiced cases, nor has it the greenish hue that we often get in long-standing cases of jaundice. It is pigment which is of a distinctly brownish color and mixed with it there is probably a certain amount of jaundice, but, as you look at the hands, abdomen, and face your impression is not one of a jaundiced patient, but of an individual who is pigmented, and the pigment is, in general, of a brownish color. Her history is one of pigmentation certainly

going back about six months. She then noticed that she was yellow, and associated with that was the marked itching that we get in jaundice and which leads to a great deal of scratching. Then she says that she has always been very dark in color. She is, of course, a dark brunette, and there is a certain amount of doubt in her mind as to whether she is much more darkly pigmented now than she was in the past. She speaks of being periodically darker in color and always being pretty dark, so she is not quite sure of how long this dark pigmentation has been going on. It may date back to her early girlhood or it may be a thing that increased fairly recently, but increased gradually enough so that she herself really has not noticed any marked amount of pigmentation in the sense of change. If you did not know about that point, looking at her you would say that she was very definitely pigmented. We do not see Irish brunettes as darkly pigmented as her skin is and, as you say, that is somewhat suggestive of Addison's disease. She has a pigmented skin interrupted by non-pigmented spots, which are areas of vitiligo. Vitiligo occurs in non-pigmented people, but is particularly prominent when it occurs in pigmented people. The negro, particularly the mulatto type, very often shows vitiligo. Of course the negro has the same kind of pigment as a case of Addison's disease, except he has rather more of it, and has always had it, and the next generation is going to have it. Addison's disease is an acquired pigment, but as far as the skin is concerned there is no difference between Addison's disease in a microscopic section and the pigment in the negro.

December 12, 1918.

I want to discuss first this afternoon the diagnosis of the patient that I showed on Monday. To sum up what we found in that case, we had a woman of forty-four, who presented the picture of marked ascites with moderate edema of the legs. Her skin was deeply pigmented, brownish in color, with spots of vitiligo. In addition, there was a very slight degree of jaundice. Her abdomen had been tapped several times. After the removal of the fluid the liver and spleen could be felt and were

found to be moderately enlarged. She had a normal heart, normal lungs, and no signs of nephritis. For eight years she had had nosebleeds accompanying her menstrual periods occasionally. For two years this phenomenon had been absent with the exception of one menstrual period when she had a nosebleed, and that was her last menstrual period and, apparently, the beginning of her menopause. She was unusually well up to six months ago. Then she began to have severe itching of the skin, so much so that she scratched enough to cause excoriation of the skin. At that time she noticed that she was jaundiced. A month ago she noticed swelling of her legs, and at this time she noted swelling of her abdomen. It is not very clear from her statement whether the swelling in the abdomen antedated the swelling of the legs or not; apparently she noticed the swelling of the legs first and then noticed the enlargement of her abdomen. She feels pretty certain that swelling of the abdomen took place in a relatively short time and was not present until approximately a month ago. As to her skin color—she thinks that she has always been dark, and apparently she had not noticed particularly this dark brown color to her skin as being anything abnormal, though she had noticed six months ago that she was jaundiced. There has been a loss of weight compensated by increasing fluid, so that she has noted increasing emaciation, and yet on admission her weight was the same as it was at her best weight when she was well. There was no history of use of alcohol and no venereal disease by history, symptoms or signs, and her blood Wassermann reaction was negative.

That case raises the question of the nature of the hepatic lesion, because the evidence is pretty clear that there is a disturbance in the liver, and the question of the nature of the pigmentation. It seems pretty clear that the change in the liver is of the nature of a cirrhosis, possibly associated with obstruction in her common bile-duct, that the ascites is secondary to the portal obstruction, that the swelling of the legs is due to the accumulation of fluid in the abdomen pressing upon the inferior vena cava or some of its intra-abdominal branches. The enlargement of the spleen may be the result of the portal

obstruction or may be due to some process of hyperplastic nature going along with the change in the liver. That the latter is the case is suggested by the fact that the spleen is of considerable size, when apparently from her symptoms the portal obstruction has been of short duration. As far as the pigment in the skin is concerned vitiligo is probably an accidental accompaniment and has no significance. Six months ago she had itching and noticed jaundice. Apparently the intensity of the jaundice was not very great. The itching, however, accompanying the jaundice was sufficient to cause vigorous scratching and suggests that at that time there undoubtedly was a disturbance of the nature of jaundice because itching is a very common accompaniment of jaundice, though it does not run parallel in amount to the jaundice. Some of the patients with the most marked itching have very little jaundice, and some of the patients with very marked jaundice have very little or no itching of the skin. In other words, pruritis is a common accompaniment of jaundice, but does not occur directly in proportion to either the extent or the duration of the jaundice. She was probably pigmented, from her story, in addition to the jaundice at the time of the development of the jaundice and the itching. In other words, she had a brownish pigmentation at that time and possibly has had it for a long time. At the same time she is abnormally deeply pigmented for a woman of her race, she being an Irish woman. Although she is a brunette, you would not expect her skin to be as dark as it is unless there were some pathologic condition.

What conditions cause a combination of cirrhosis with enlargement of the spleen and pigmentation of the skin? The condition that you have to think of with her associated symptoms is hemochromatosis. Hemochromatosis is a condition in which there is an abnormal deposition of iron-containing pigment in the connective-tissue cells of various parts of the body, in the skin causing discoloration, in the liver causing, or at least associated with, connective-tissue increase and the pathologic picture of a chronic interstitial hepatitis, in the spleen associated with hyperplasia, possibly causing hyperplasia, in the pancreas

causing interstitial pancreatitis with connective tissue increase, and in the pancreas usually deposited also in the cells of the islands of Langerhans, and associated with these changes disturbance of carbohydrate metabolism leading in the large majority of cases to actual diabetes, the so-called bronzed diabetes.

This particular patient of ours in all urine examinations has shown a very slight amount of glucose in the urine; in other words, a slight degree of glycosuria, present in small amount when the patient is not on a carbohydrate-limited diet.

The pigment of hemochromatosis is also deposited in other organs of the body, though in other organs of the body it rarely ever causes enough change to lead to symptoms. The pigmentation is increased in amount in the heart muscle and yet the heart usually functions normally. It is increased in the kidney, though there is usually no connective tissue increase in the kidney and nothing to suggest chronic interstitial nephritis as a result of the deposition of the pigment. It is increased in the striated muscle of the body in general and increased in the smooth muscle. Very often there is a considerable deposition in the smooth muscle of the corium.

So much for the general picture of hemochromatosis. There is a good deal in what I have said in summing up this case that is in agreement with hemochromatosis. Against hemochromatosis in this particular case is the history of jaundice occurring six months ago, apparently persisting for some time, as indicated by the itching, and present prior to the development of the ascites. Her ascites is against hemochromatosis because most of the cases of hemochromatosis whereas they have a cirrhotic change in the liver do not have portal obstruction and ascites. The sex of the patient is in a sense against hemochromatosis because of a very considerable number of cases reported in literature nearly all have occurred in males. I believe, in a group of cases which Futcher reported of 36 patients, 2 were females and 34 males. Of course this may be the unusual case, and the relative infrequency in females of the disease is no justification for excluding that diagnosis in this individual case.

Does the glycosuria necessarily indicate a lesion in the pan-

creas presumably due to the deposition of the pigment, and is that part of the picture of hemochromatosis? Usually cases of hemochromatosis when they develop glycosuria have glycosuria of the extent and character of the average diabetic, glycosuria considerable in amount when the patient is on a diet with an average amount of carbohydrate, and frequently a glycosuria which persists even with a great reduction of carbohydrate intake just as you get in diabetes. However, typical hemochromatosis with typical lesions in the pancreas may show only a slight glycosuria, may show a transient glycosuria, or may show no glycosuria at all. We have observed cases in this hospital which were proved at autopsy to be typical cases of hemochromatosis in which glycosuria was a very trifling and transient symptom over a very considerable observation in the hospital with the patient on a free carbohydrate diet.

The ascitic fluid of a case of cirrhosis very frequently contains a small amount of glucose, so that you get a positive Fehling test. I believe in this particular case there was no glucose in the ascitic fluid, but the finding of glucose in ascitic fluid is not at all a surprising accompaniment of ascites from cirrhosis of the liver. A slight degree of glycosuria is very common in chronic conditions where there is no evidence of diabetes. Such a condition is represented by hypertension with slight renal lesion. A good number of our cases of hypertension show slight degrees of glycosuria. Slight degrees of glycosuria are not uncommon in simple cases of chronic cirrhosis without any pigmentation, so that one factor, though suggesting hemochromatosis, is not definite.

The pigmentation in itself in this particular case is rather more of a brownish color than you usually get in hemochromatosis. Hemochromatosis is much more apt to have a reddish hue. Here is a Lumière plate (Fig. 235) of one of our earlier cases (Med. No. 3957), a man who remained in the hospital for the better part of two and one-half years, and you will see there distinctly the reddish or copper-red color. This man, like the present patient, was of Irish extraction, had the Irish sense of humor, and rather liked to boast of the fact that he was the only living

Irish Indian. He did have that sort of copper-reddish brown color that is characteristic of the Indian. This pigment then in itself as far as color goes is not characteristic of hemochromatosis, though I have seen cases of hemochromatosis in which the discoloration was quite similar to this particular case.

What is the cause of hemochromatosis? Is there anything in the nature of the lesion that will give us help in making a diagnosis? The type of pigmentation is, as I have already described, a diffuse one. If you are interested in the pathology there is a very good pathologic discussion of hemochromatosis in the Archives of Internal Medicine, 1911, vii, 75, by Sprunt, with photographs showing the distribution in the tissues of the pigment and a discussion of the pathogenesis of the disease. After reading the various discussions of the disease, as outlined by Sprunt, you can sum it up as to etiology by saying that Dr. Sprunt and the rest of us do not know very much about it. That article was written in 1911. Since then there has been experimental work that throws some light on the possible mechanism of the production of the condition. In a paper published in November of this year in the Journal of Experimental Medicine (1918, xxviii, 629) Dr. Rous and Dr. Oliver reported the production in animals of what they called experimental hemochromatosis. It was produced in rabbits in which animal plethora had been produced by repeated injections of blood. These injections of blood were carried out in connection with some other experiments in which they were studying the fate of red blood-corpuscles when injected into animals. They noticed that their experimental animals showed pigmentation, and the pigmentation in character and distribution was quite similar to the pigmentation of hemochromatosis. Then they found, like other observers, that there was a certain amount of pigment deposited in the kidney, and they observed in the urine the appearances of cells containing pigment, and that suggested in their minds that this might be a possible diagnostic help in hemochromatosis. Subsequently they had the opportunity to study the urine of a case of hemochromatosis in the hospital, and they found there pigmented cells in the urine, and as a re-

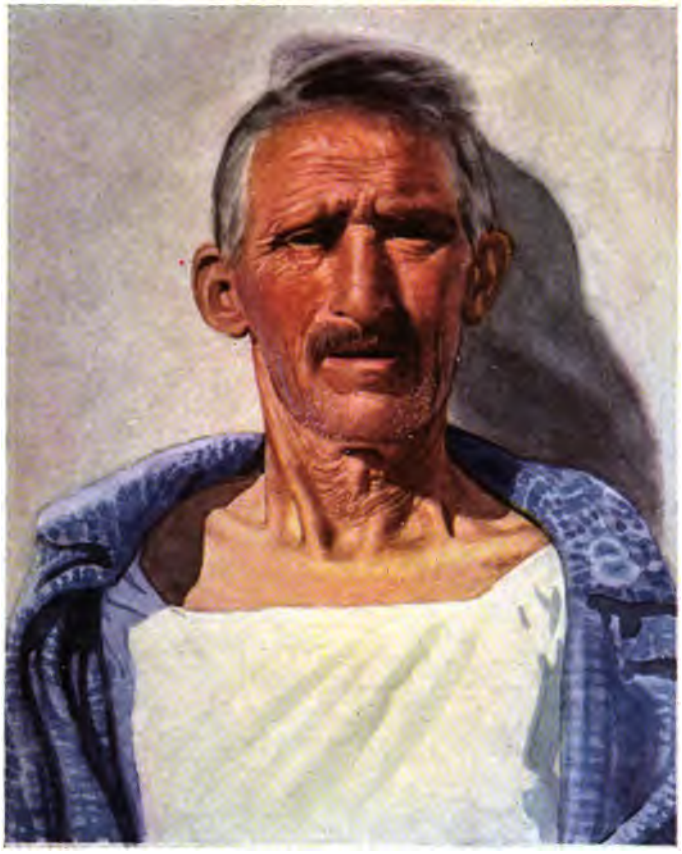

FIG. 235.—A case of true hemochromatosis referred to in comparison with the patient discussed in the clinic.

sult of that Dr. Rous in the same journal published another paper entitled "Urinary Siderosis," with pictures of the kidney and the pigmented cells in the urine (Jour. Exp. Med., 1918, xxviii, 645). That case subsequently died, and at autopsy it was seen to be a case of hemochromatosis. That work suggests that the cause of the disease is some destructive process in the red cells leading to breaking up of the red cells at an unusual rate with deposition of the pigment, set free in that breaking up, in various connective-tissue cells of the body, with possibly a certain amount of metabolic change in the pigment, leading to the deposition of hematoidin, a by-product of hemoglobin, not possessing the pigmentary characteristics of hemoglobin, but a pigment containing iron, which will give the test for iron when the proper stains are used. If Rous' idea is true then cases of hemochromatosis should be anemic unless blood regeneration is active enough to keep pace with blood destruction. If blood regeneration is active enough to keep pace with blood destruction and prevent anemia, then at autopsy these cases should show signs of hyperplasia in the structures that form red blood-cells, namely, in the bone-marrow. Our patient showed, as I said, very little anemia. I gave you her hemoglobin, which is very little below normal, and this is true of most cases, and anemia is not a common accompaniment of hemochromatosis, and our case agrees in that particular with typical cases of hemochromatosis. These cases do not suggest having had a plethora of blood. Also it is true that the bone-marrow in this case is not likely to be a hyperplastic bone-marrow; as there is no evidence of active blood regeneration this explanation of Rous may not fit in with the actual conditions in the human cases, though it is suggestive.

Another view, which was advanced first, I think, by Meltzer some years ago, without very much proof for it, was that the pigmentation is not due to destruction of red cells, is not associated with any form of potential or real anemia, but is due to disturbance in the metabolism, which leads to a storing of iron in the body because the ingested iron which is present in much of the food instead of being excreted is converted into some

form not easily excreted, and after absorption is deposited in the tissues. Recently there have been some studies to see whether in the human being with hemochromatosis there is a disturbance in the iron metabolism. Dr. Stevens, who was for a short time Assistant Resident in this hospital, with Dr. Campbell Howard of Iowa City, studied the metabolism in a human case, and they published their results in the Archives of Internal Medicine, 1917, xx, 896, and they gave what the literature had on the question of iron metabolism prior to that time. They studied a case rather late in the course of the disease. They found evidence of a slight storing of food iron without any signs of undue destruction of blood-cells. Their method was to put the patient on a fixed diet, which the patient could take daily for a period of six to seven days. They studied the urine and studied the feces during that time, quantitating the iron in the intake of food and quantitating the iron in the output of feces and urine. The striking thing about their observations is that the urine chemically showed no iron. The iron that was excreted was excreted by the gastro-intestinal tract, was found in the stools, but was not found in the urine. You will still find on page 899 of that article a table of intake of iron in milligrams and the output in the urine. The patient was studied for five weeks in this way. Their findings you see are not exactly in accord with the observations of Rous, who found iron in the urine, unless it is explained by the fact that the iron in the urine is in the cells and that the cells were sedimented out, and they chemically quantitated the iron in the urine after the sedimentation had taken place. That is not at all improbable, because these are rather slow and tedious chemical methods, and I have no doubt that the urine stood for some time in the ice-box while other work was going on, and the sediment may have gone to the bottom and not been quantitated.

Dr. McClure published in the Archives of Internal Medicine for November, (1918, xxii, 610) a similar metabolic study carried out here in this hospital in one of our cases of hemochromatosis, with essentially the same result, the finding of a stored amount of iron, the food intake of iron being greater than the

iron recovered in the stools, none being found in the urine. If you look up those figures you will find that the amounts of iron are pretty small, and it is not at all improbable that the discrepancy between the intake and the output, which is a small one, may be due to errors in technic and methods of observations, but both studies agree in finding a stored amount of iron, and they are suggestive of that mechanism of the production of the lesion, putting the disease in the group of metabolic disturbances and a disturbance in the metabolism of iron. Unfortunately, there have not been any very extensive studies, under normal conditions or in other diseases, of iron metabolism, and there is not any very satisfactory data with which to compare these studies. Until that is done I do not think that we can feel too sure that there is an actual disturbance of the iron metabolism.

This particular patient has not had an iron metabolism study. We have excised a piece of skin, and that skin shows a fairly definite increase in the pigment in the lower layer of the epithelium. You remember that in the lower layers of the epithelium of the skin there are cuboidal cells, and then the cells become more and more flattened out in the upper layers until you get to the horny layer. Normally in those lower cells there is a deposition of a varying amount of pigment, always present in the skin, more abundant in dark-colored people than in people with a light-colored skin. In this case there is more than the normal amount of pigment in that layer and in some of the upper cells, and that pigment under the microscope is seen to be made up of rather coarse granules of brownish or, better, yellowish color. In the subcutis are single connective-tissue cells with coarse granules of pigment of the same type pretty thickly studding the cell. There is not any particular amount of pigment around the sebaceous glands or sweat-glands, and no increased pigment in the smooth muscle of the deeper layers of the skin. These connective-tissue cells are to be regarded as chromatophores, probably cells normally pigmented. Whether that pigment is iron containing or not I will have to report later, because the first piece of skin excised was

hardened in Zenker's fluid, and stains to demonstrate iron do not work very satisfactorily after Zenker fixation. There is a bit hardened in alcohol going through now, but the Pathological Department has not completed the staining.

We have examined the urine of this patient to see if any pigmented cells were present in the urine that gave the iron reaction, and we found that there was a certain amount of iron-containing pigment, but we were skeptical, as we should be, and I suggested to Dr. Balyeat, who made the test, that it was very important to take the urine from somebody else supposedly normal and carry out the same procedure, and he found about the same amount of apparently iron-containing pigment in the normal individual. What its source is I do not know. Some of the cells from the urine of this patient and I think from the ascitic fluid have been run through and stained for iron, and there are pigmented cells in both places which seem to show the iron reaction.

Now we have the evidence of pigment in the skin, have some evidence of iron-containing pigment in the ascitic fluid and in the urine from this case, but I am still quite skeptical as to whether that represents a diagnostic point that justifies us in saying that this is a case of hemochromatosis or not. You have to attach a good deal of importance, I think, in this particular case to the fact that this woman is intelligent, that she has given an exceedingly good story of the onset of her symptoms, and apparently her observations have been accurate. She herself has not noticed any rapid change in her skin, she thinks that she has been pigmented for a long time, probably ever since she was a young girl. Then we have that history of jaundice and itching, which would agree with the diagnosis of some biliary disturbance causing jaundice, and, with the itching of the skin, of having to scratch a good deal. That irritation may have led to pigmentation, but I feel skeptical from her history of scratching, etc., that it was sufficient to cause an increase in pigmentation, though, if already there was a pretty marked pigmentation, it might be accentuated as a result of scratching. It is well-known that irritation of various kinds

from the pruritus of jaundice to the itching from lice leads to scratching, and that scratching leads to pigmentation. The vagabond is often a pigmented fellow after you get the dirt off and can see the skin. Then there is a variety of pathologic conditions causing increased pigment varying all the way from chronic ascites to solid ovarian tumors, tubercular peritonitis, etc. Another of the causes of pigmentation is a chronic renal condition. These cases of chronic jaundice with itching sometimes become very much pigmented, and you have a great deal of difficulty in a number of cases in determining whether the pigment is abnormal in the sense of hemochromatosis or whether the pigment represents an extension of the normal pigment of the skin due to various irritating causes, or whether the pigment is really of biliary origin.

On the whole, I am a little inclined to think that this patient represents a case of simple cirrhosis of the liver with ascites in a woman pretty dark to start with, and her pigment accentuated by scratching and with possibly an added factor in a chronic irritation in the peritoneal cavity in the nature of ascites, such as we find in solid ovarian tumors, etc. An association of the pigmentation with changes in the adrenal has not been excluded, but the case is not similar to the usual case of Addison's disease.

Hemochromatosis is rather a rare condition. For instance, up to 1912 Potter and Milne could find only 50 cases of bronzed diabetes, that is, hemochromatosis with diabetes. Since then, of course, a good many cases have been added to the literature. That paper in the American Journal of Medical Sciences (1912, cxlii, 46), by Potter and Milne, gives all the reported cases up to that time and discusses them, and is a useful summary of the literature.

January 20, 1919.

One of the men asked in regard to the findings in the skin in that patient (Med. No. 9841) that I showed you some time before Christmas with marked enlargement of the liver, etc., where there was a question of hemochromatosis. The skin shows an increased amount of pigmentation, and that pigmentation so far as the pathologists have been able to determine is not

an iron-containing pigment or a pigment that gives the iron reaction. There is a considerable amount of pigment in the lowest layer of the epithelium, and that looks just like the normal pigment of pigmented skin such as you get in the negro, or the kind of pigment that you get following an irritation, such as sunburn. Then the connective-tissue cells in the skin have more than the normal amount of pigment, and that pigment is fairly coarsely granular, brownish-yellow in color under the microscope, and is deposited in practically all of the connective-tissue cells. Apparently no pigment was found in the smooth muscle. I compared that pigment this morning with sections from a previous case known to be hemochromatosis, and the pigment is in slightly smaller granules, but otherwise looks pretty much the same; but so far as that study goes there is no positive, definite evidence that that is the kind of pigment we get in hemochromatosis. On the other hand, it may be an iron-containing pigment in which technically we failed to demonstrate iron, or it may be a case of hemochromatosis and the skin pigment not contain iron. Such cases are reported.

The patient has remained in the hospital, and my impression is that in an interval of about three weeks when I was away from Boston and did not see her that the pigmentation has increased. It looks like a darker pigmentation. That is merely an impression; we have used no accurate way of measuring such changes. There has been no other change in her condition except pain in her abdomen. This has increased and radiates now down her legs. She continues to show a trace of sugar in her urine. The findings are perfectly consistent with hemochromatosis, but we have no positive proof that it is hemochromatosis, and I am inclined to think it is not hemochromatosis.

February 19, 1919.

I want to report on a case which has not changed much since you saw her—the woman (Med. No. 9841) with the marked pigmentation, moderate enlargement of the liver and spleen, in which we raised the question of her having hemochromatosis, but in the skin of whom could not be demonstrated any iron in

the pigment. The patient has not changed very much in general appearance, but there is a question as to whether the pigment has not moderately deepened. From time to time she fills up with fluid and requires that her abdomen be tapped. Following the tapping she now has a considerable amount of pain, and all the time she has a sort of constant pain in her abdomen which tends to radiate down her legs, and is severe enough so that she has to have some opiate to make her comfortable. I do not think that at the time we had her in the clinic that she was complaining very much from pain or suffering any great amount of discomfort, except simply discomfort from the distention of the abdomen, so that feature has been added, and the nature of the pain radiating down the legs suggests some form of pressure on nerves, and in that way the pain referred down the legs. After she is tapped the abdomen feels just as it did before; you can feel the liver edge beneath the costal margin and you can feel the spleen two or three finger-breadths down on the other side. There is now pretty definite spasticity of the lateral abdominal muscles, but no spasm and no definite mass of any kind can be felt in the abdomen beyond the liver and the spleen. You remember that that patient gave also the history of jaundice. She shows a slight degree of jaundice now and she has bile in her urine. Her stools are clay colored. There is nothing definite to be added in the way of a diagnosis further than to say that as it progresses it has not become any more clearly a case of hemochromatosis, and the pain is somewhat suggestive that there may be something like a neoplasm in the retroperitoneal tissues, but there is nothing positive of that. There is evidence of portal obstruction causing ascites and evidence of an intrahepatic disturbance causing the jaundice and of obstruction to the flow of bile into the intestine. As far as that goes, it fits in with the idea that it is a case of cirrhosis, possibly with a stone in the common duct. Of course, it would be cirrhosis if it were a case of hemochromatosis. The iron pigment in hemochromatosis is deposited in the increased connective tissue in the liver. There has been no change as regards the glycosuria. It is still present in small

amount, but there is not a definite diabetes. I will report later on that patient as to any changes. She is still on the ward.

March 17, 1919.

This (P. B. B. H., Med. No. 9841) is the patient that I showed in the clinic of December 9th and 12th, and reported about on January 20th, and again on February 19th. She has been in the hospital a total of one hundred and twenty-four days. Her abdomen has been tapped twelve times, and from 6 to 7 liters of fluid removed each time. There has been no change as regards the glycosuria. Her general condition has remained as described at the last report. She has had this persistent discomfort which requires an opiate. Sometimes following the tapping she has had a very considerable degree of pain in her abdomen which decreases after she begins to fill up again, but it never disappears. Following one tapping one of the house-officers thought that he could make out a mass, which was irregular and firm, in the retroperitoneal region of the epigastrium. Following previous and subsequent tappings nothing definite of this nature had been made out, and it seems rather probable that what was made out was the liver and not some other mass. She has continued to have bile in her urine, and you can see, I am sure, that now she is pretty deeply jaundiced. Her conjunctivæ are definitely yellow. In places in the brownish color she has, as she had when you saw her several months ago, areas of vitiligo. The abdomen shows more of the brown color than her face and extremities, as it did before, and with this there is intermixed a certain amount of yellowish tinting, and these patches of vitiligo show some slight yellowish color; in other words, where the brown is not so dark there is quite evident jaundice. She has persistently had clay-colored stools, and associated with that there has recently been some diarrhea. The stools have not now for a long time shown any color indicating bile, and such bile tests as have been made have been negative. You probably recall that early in her stay in the hospital the stools were not clay colored, and a little later, though the stools were very pale, bile was demonstrated in them. As she has progressed in her illness jaundice has become much

more an evident feature in the case. There was a history of jaundice in the beginning, and very slight jaundice was present when she came into the hospital. That jaundice has become more marked and the element of obstruction has become more marked; that is, there has been no bile in her stools while her jaundice has been increasing, so that, as it stands today, just what I said in regard to the diagnosis before, I think, holds good. There is evidence of an intrahepatic disturbance causing the enlargement of the liver; in other words, a form of hepatitis; the spleen is enlarged, splenitis, and associated with that there is jaundice and also evidence of biliary obstruction. Though you cannot say just what the biliary obstruction is due to, the whole history is rather more suggestive that the biliary obstruction is due to gall-stones than that it is due to neoplasm obstructing the common duct. There is not any history of pain, there is no recurring fever, as you often get with stone in the common duct, and, as I have repeatedly said, there is no definite evidence to exclude a neoplasm. In the beginning the condition seemed to be one that fitted in better with an intrahepatic disturbance than a neoplasm; to this has been added definite evidence of biliary obstruction. There are no other features that have developed in the case besides the ones that I have mentioned.

March 31, 1919.

The patient (P. B. B. H. Med., No. 9841), recently shown, rapidly lost ground and died. Autopsy today showed a very marked cirrhosis of the liver with an associated chronic splenitis. The liver was a rich green in color with a nodular surface; it weighed 940 grams. The spleen weighed 400 grams. There were no stones in the biliary tract; the ducts were patent and showed no evidences of having been obstructed. The pancreas, though firm, seemed normal. The intense jaundice had resulted from intrahepatic changes and these must have caused the acholuric appearance of the stools. Judging from the fact that at autopsy bile could be squeezed from the common duct, some bile must have made its way during life into the intestine. There was nothing to suggest hemochromatosis.

FIBRINOUS BRONCHITIS

December 16, 1918.

I AM not going to ask any one of you to go over this patient (Med. No. 9990) this morning because his physical examination is relatively unimportant. The patient is twenty-two years of age, and he comes in complaining of stomach trouble and breathlessness. His family history is entirely negative. There are no diseases in his family that would suggest any hereditary relationship to his condition. He is single. His habits are good. For three years he has been a helper to a plumber. For some years previous to that he drove a milk team and went to school. There has been no history of exposure to irritating fumes.

He was born in Portland, Maine, but moved in early childhood to Boston, where he has lived since. His general health has been good. He had chickenpox, mumps, and whooping-cough. There has been no exposure to tuberculosis and no history of acute infectious diseases beyond the three mentioned. In childhood he sustained a blow in his back, which he holds responsible for a partial paralysis and shortening of his right leg. He has not been subject to head colds and has had no discharge from his nose. He has had some obstruction in his nose which I I will refer to later. There is no history of tonsillitis, sore throats, hoarseness, or sore mouth. His cardiorespiratory history is negative.

He has always eaten generously of a wide variety of food, and has had an excellent appetite. His bowels have always been a little sluggish, so that once in two weeks he has had to take a cathartic. There has been no nausea, vomiting, gas, hematemesis, distress, colic, icterus, diarrhea, clay-colored, bloody, or tarry stools. No hemorrhoids. His genito-urinary history is entirely negative. His neuromuscular history is entirely negative. His best weight was 165 pounds six months ago. Recently he has weighed 127 pounds. His loss of weight has been entirely

during his present illness, which has been of three months' duration. His present illness began three months ago.

The patient was awakened three months ago by a severe cough and feeling of breathlessness. The cough was succeeded by the eructation of much gas, during which time his breathlessness and feeling of oppression grew steadily worse. After an hour the attack gradually passed and he went back to sleep. Each night since he has had similar attacks, increasing in frequency, duration, and severity. The attack almost invariably begins with a cough, which raises a varying amount of sputum, usually thick and greenish, occasionally white and frothy. The cough is followed by the distention with and eructation of large quantities of gas, which is succeeded by difficult and wheezy breathing. Expiration is much easier than inspiration. Often the only way to relieve his distress is to take up a position on his hands and knees. Toward the end of the attack vomiting frequently occurs. The vomitus is green and watery and never contains food.

He describes his sensation during the attack as though a hand were squeezing his windpipe, and the end of the attack comes when the hand gradually lets up. The attacks occur rarely in the daytime, although he is frequently wheezy and inclined to coughing spells. After meals is his most dangerous time of the day, but these attacks are less severe than the inevitable nocturnal attacks. The cough makes his head ache and often brings on vomiting. Two months ago his increasing weakness forced him to stop work. Five weeks ago he went to a local doctor, who discovered a polyp in his nose, and is said to have removed it. His nasal obstruction was partially relieved, but his attacks were no fewer.

About a month ago he went to a doctor in Portland, who gave him adrenalin hypodermically, and this relieved his attacks promptly. Every night since then he has had to have adrenalin sometimes several times during the night. It always gives him temporary relief, but he thinks he is apt to be more wheezy next day. He has vomited almost every day of the past fortnight, always after coughing. Solid food makes him so much worse

that for weeks he has lived on milk and milk-toast. Coughing gives him a severe headache and some epigastric pain. He also complains that a deep breath hurts his stomach. A few times severe coughing has led to faintness. He is increasingly constipated. He complains of a nasty taste in his mouth.

His physical examination, as I said, shows relatively little. In regard to his pulmonary condition, when he came in, this note was made: "Over the whole of both lungs, most marked near their bases, are heard multitudes of squeaks and groans, and occasional coarse moist râles. All are more marked on inspiration. Resonance is unimpaired. Tactile fremitus, voice, and breath sounds are quite normal. Lower borders of lung in back are low and move very slightly with forced respiration."

The morning after admission the physical examination was perfectly negative. Three or four days later I made this note on his lungs: "The lungs are slightly hyperresonant throughout. Expiration is distinctly prolonged and slightly high pitched. Apparently normal pulmonary condition."

Since he has been in the hospital he has run a little fever, the highest being just a trifle over 100° F. His respiration has not been particularly rapid. His pulse, at times, has been a little rapid. Practically every night he has had an "asthmatic" attack, and several times during the day he has been moderately dyspneic. In the first severe attack he had in the hospital the difficulty seemed to be inspiratory chiefly. Inspiration was quick and deep and expiration much prolonged and wheezing. Adrenalin relieves his attacks promptly, but makes him tremble and makes his eyes burn. A very mild attack, a day or two ago, was relieved, as it seemed, by a sterile hypodermic, but last night he had a very severe attack on which a sterile hypodermic had absolutely no effect, though his attack was relieved promptly by adrenalin.

On December 13th this note was made: "Patient's asthmatic attacks have continued, but are promptly relieved by adrenalin. He has had at least one attack every night since entry. He is running a slight temperature between normal and 100° F. Yesterday he was started on a saturated solution of potassium

iodid, 1 c.c. three times a day. Dr. Walker is giving him sputum vaccine."

DR. CHRISTIAN (to Patient): How about your attacks in the last two or three days?

PATIENT: I have only had one since Dr. Walker injected the serum.

DR. CHRISTIAN (to Patient): What day was that?

PATIENT: Last Thursday. (Clinic on Monday.)

DR. CHRISTIAN: On last Thursday he had an injection of "serum." A vaccine has been made from the organisms from his sputum, and he has had only one attack of dyspnea since that was given. Also he has been receiving a saturated solution of potassium iodid, 1 c.c. three times a day. Here is a specimen of his sputum which I will pass around. It happens that, since he has had this treatment, he has not had in his sputum what I am particularly anxious for you to see, namely, bronchial casts. He coughed up in his early stay in the hospital casts of his bronchi which showed branching, with various subdivisions going down into finer ramifications, the kind of thing that is shown in this picture that I will pass around [1] (Fig. 236). I think you can see it is simply a cast of the bronchial tree. Then his sputum has been of the watery character, a considerable amount of sputum, and in it floating masses, flocculent masses, and, when he coughs up these casts, they come up, as you might expect, in just such flocculent masses, but in the fluid they separate out and you can see the casts. Examined under the microscope the casts have the appearance of a homogeneous matrix in which are running fine fibrils or threads, and covering the casts or mixed in with the casts are a considerable number of leukocytes with rather coarse granules. These are eosinophilic cells.

(House-officer asked if any Charcot-Leyden crystals have been found in this case.)

ANSWER: No, none have been found.

DR. CHRISTIAN: None have been found in his sputum, so he has that type of sputum with these casts of his bronchi and with

[1] Illustration from Bettmann's paper, Amer. Jour. Med. Sci., 1902, cxxiii, 304.

eosinophilic cells. He has no eosinophilia in his circulating blood. His differential blood count is normal. He has moderate leukocytosis, 15,000 when he came in, which gradually dropped to 8100.

His pulmonary examination shows wheezing and at times bronchi. *x*-Ray plate shows no areas of consolidation, but shows rather a marked bronchial tree, rather more than we see in the average chest plate. You see it well marked here (indication to

FIG. 236.—Fibrinous bronchitis cast expectorated; three-fourths natural size (Milton Bettmann, in American Journal of the Medical Sciences).

plate), and you see the ramification going to the lower lobe and also to the upper lobe. Here is a small round dense area, possibly suggesting a small area of calcification. At the roots of the lungs on both sides there is a good deal of density, either thickening around the bronchi or some enlarged peribronchial lymph-nodes. There is no evidence in the plate of infiltration at the apex or anywhere running out to the periphery of the lungs; so,

as far as the process goes, it is suggestive of peribronchitis with possibly some dilatation of the bronchi, but that is not very evident. It is more a plate of peribronchitis than bronchiectasis. There is no deformity of the diaphragm and no signs of pleural thickening.

Occasionally in these cases you can get queer signs on auscultation described as flapping or crackling sounds due to the air being pushed by the cast in the bronchus, and the bronchus acting with the cast in it temporarily with a sort of valve-like action. All he shows this morning is the rather louder prolonged inspiration, and with expiration there is a certain amount of crackling sound, no particular amount of râles, and no real extraneous sounds. Sometimes when you get an extraneous sound it serves to locate what part of the pulmonary tissue the cast comes from. The effort has also been made to locate the origin of the cast as to whether it is from the upper or lower lobe, because casts from the upper lobe do not have as many or varying branches as those from the lower lobe, but that diagnosis would be possible only with large casts and pretty numerous ones.

This is distinctly an unusual condition. It is what is spoken of as fibrinous bronchitis, and Babcock in his book has given it the term "plastic bronchitis." The reason for calling it plastic bronchitis rather than fibrinous bronchitis is that, taking up the question of the structure of these casts, they often do not contain very much fibrin, some are nearly entirely made up of mucus, and in others there is a considerable amount of fibrin, but in most cases there is a mixture of both, and the term "plastic" does away with the difficulty of the term "fibrinous" when the thing is not made up of fibrin.

Here I want to read just briefly the history of two other cases to impress on your minds the characteristics. The following was a patient of Dr. Landis,[1] of Philadelphia: "The patient was a robust healthy male, aged forty-seven, referred to him because of suspected tuberculosis. The family history was negative and he had never had any illness. Four weeks prior to his coming

[1] "Diseases of the Chest and the Principles of Physical Diagnosis," Norris and Landis, W. B. Saunders Company, 1917, page 265.

under observation he had caught cold. The trouble started as a coryza, and in a few days had developed dyspnea. For the first two weeks the sputum, which was moderate in amount, was thick and whitish in color, later it had a greenish tinge." (Remember, our patient's description of the sputum was that it was whitish and sometimes greenish.) "On one occasion after a paroxysm of coughing he spat into his handkerchief a small hard pellet, which, on examination, proved to be a cast from one of the medium-sized bronchi. There was a slight elevation of temperature during that attack. The sputum was negative for tubercle bacilli, but contained both pneumococci and streptococci. With the exception of one day spent in bed he attended to his professional work, although with some effort. His appetite was good and he had lost no weight. On physical examination there was deficient expansion at the right base, impairment of the percussion note, suppressed breath sounds, both large and small râles, and, in addition, a flapping sound heard at the end of inspiration. The patient was sent to the seashore and, when seen two weeks later, there was nothing abnormal to be noted over the areas previously affected."

Another case is one reported by Dr. Bettmann in the American Journal of Medical Sciences for 1902, cxxiii, 304: "The patient was a negress aged twenty-two years, who came to the hospital on the obstetrical service. She gave a history that six years before, in the fall, she had an attack similar to the attack observed in the hospital, the symptoms being cough, pain, respiratory distress, profuse expectoration, containing, evidently from the patient's description, branching casts. Following this attack she had pneumonia and was confined to bed for one month. Every year in the fall since that time she has had similar attacks. These attacks had no relationship to her previous pregnancies, which occurred two and three years previous to entrance to the hospital, respectively. She has lost weight throughout the last two years." (Most of the rest refers to her pregnancy, which was normal, with a normal delivery, etc.)

"On the tenth day following her delivery she was allowed to sit up in a chair, but the discomfort from her cough increased so

much that she was put to bed again, her evening temperature having touched 102° F. On the thirteenth day of her puerperium I was in the ward delivering another case, and my attention was called by the nurse to the patient's extreme dyspnea." The description of the dyspnea is as follows: "The patient is sitting up in bed in intense dyspnea. All the accessory muscles of respiration are called into play. The alæ nasæ dilate with each respiratory movement. She coughs incessantly, and brings up large masses of expectoration. She is complaining of an intense sharp pain radiating from the lower left side through the back, increased on coughing. Respirations are 35 per minute; pulse 100; temperature 102° F. Loose tracheal râles are heard in the throat. On palpation there is normal vocal fremitus throughout. On percussion there is high tympany in both lower axillary regions, higher and approaching dull on the right. On auscultation over the front respiration is prolonged and accompanied by moist râles. The breathing is more intense in the right lower axilla, and approaches tubular breathing in its quality. The inspirations are here accompanied by râles distincly crepitant in character. The attack of dyspnea lasted about one hour. That attack was on September 25th.

"She had another attack not so severe in character on October 11th, eleven days later, and between those two attacks she had slight respiratory distress and her evening temperature ranged between 100° and 101° F. She had slight cough and occasionally expectorated small casts, which caused her very little discomfort. On one occasion a to-and-fro vibrating sound was heard on expiration in the right axilla. This corresponds in character to the 'ventilgerausch' of the Germans. The urine was negative throughout. The patient continued to have a slight cough and dyspnea and slight evening temperature, and left the hospital in spite of protests October 10th. She remained in Baltimore three weeks after her discharge from the hospital, and had frequent attacks of violent coughing and expectorated 'tree-like things,' according to the description of a friend. She then went to her home in Virginia, where she died two weeks later. No data as to the cause of her death were obtainable."

In this paper by Bettmann there is a very careful review of the literature and a classification of the cases, and he subdivides the cases into nine different groups: (1) Chronic bronchitis with expectoration of branching casts of the bronchial tree, 27 cases. Those are all the cases he could find reported in the literature up to 1902. That is what happened, judging from the description, in his case and also in the case of Dr. Landis, and they very closely resemble our case. I should call it chronic bronchitis with expectoration of branching casts of the bronchial tree. (2) Acute bronchitis with expectoration of branching casts of the bronchial tree, 15 cases. In those acute cases the attacks come on more suddenly, are of shorter duration, and are accompanied by a higher range of temperature. In those cases the temperature sometimes goes up to 104° F.; in other words, it is more like acute bronchitis, but it is rather difficult, I should think, in classifying them to say just which cases go into one group and which in the other, that is, they shade into each other. (3) Six cases in which no casts were found during life, but were an accidental finding after death. (4) Cases in which the casts expectorated showed no dichotomous branching, 11 in the literature. (5) Expectoration of branching casts in organic heart disease, 10. (6) Expectoration of branching casts in pulmonary tuberculosis, 14. (7) Expectoration of small casts, often non-branching, in association with asthma, 5. (8) Formation of casts in the bronchi in association with pulmonary edema following thoracentesis, 4. (9) Cases whose classification is doubtful because of incomplete reports, 6. You see the total number of cases from the literature is a relatively small one. The association with tuberculosis is of interest. This case of Dr. Landis was sent to him with the probable diagnosis of tuberculosis, but he could not find any evidence that there was tuberculosis. Dr. Landis says that he has seen no evidence of bronchial casts in the ward patients in the Phipps Institute, which is a tuberculosis hospital, over a period of thirteen years (he is Physician-in-Chief to the hospital), nor have such casts ever been found in any of the 662 autopsies on tuberculous patients, which brings out the probability that the finding of the condition in a patient with tuberculosis is simply a coincidence. It just

happens to occur in a case of tuberculosis. The other point of view is that the tubercular lesion is responsible. The same remark is applicable to the finding of the casts in chronic heart disease. It may be that it is simply an accidental association and not a causal relation.

In regard to the association with asthma, it brings up the question as to whether, after all, it is not a mistaken diagnosis to call these cases of bronchial asthma. We have had under observation here somewhere between 400 and 500 cases of bronchial asthma, and this is the first patient with a history suggestive of asthma in which we found these casts. Of course, Curschmann spirals, of large enough size to be picked out macroscopically, are very common in asthma, but Dr. Walker has never seen these branching casts in his asthmatics, and both Dr. Walker and myself remarked on seeing this history that it was not the history of an asthmatic. This patient has a different kind of dyspnea. The difficulty is in inspiration rather than expiration, and asthmatics do not get on their hands and knees to relieve their dyspnea; they go and sit by an open window. The asthmatic very often, with his paroxysms of asthma, has an associated gaseous distention of his stomach, probably due to the swallowing of air during his asthmatic attack. Is it not uncommon for the typical asthmatic to come in complaining of his stomach, and to tell you that, accompanying or following an asthma paroxysm, he belches up a large amount of gas, and very frequently they say that they relieved their asthma attack by the belching of gas. Some say they have indigestion and gaseous distention preceding the asthmatic attack, so that phase of the thing in this patient is not different from asthma. Very often the asthmatic attack with strain, etc., does lead to vomiting, but, taking the story of this patient's attacks as a whole, it is not the definite picture of asthma. Dr. Walker made a note the day after the patient came in, "No evidence of bronchial asthma in this case at the present time."

The etiology of the condition is quite obscure. There are one or two postmortem observations that show no definite lesions in the lungs. In cases where the casts have been found, the

bronchi may show a break in the continuity of the epithelium. In other cases the bronchial epithelium has been seen to show large numbers of mucus-secreting cells, and they have been very prominent, as if there was an exaggerated secretion from the normal mucous glands of the bronchial mucosa.

The structure of the casts have been studied in various cases. I will pass this article of Dr. Bettman (loc. cit.) around because there is a picture on page 306 of the microscopic appearance of a section of one of these casts, and on page 309 there is a macroscopic drawing of a cross-section of a large cast. The structure in the larger portion is partly tubular, that is, the cast does not fill the larger bronchus with a solid mass, but is like a deposit on the surface of the mucous membrane of varying thickness, and so the central part is incomplete. In other words, it is a tubular cast rather than a solid one. If you study that a little more carefully, you will find that, instead of being on cross-section simply a more or less circular mass with a smooth inner wall, you will find that inside the outer layer are whirls and masses of various size as represented in that picture. This suggests the mechanism of formation of a cast as being formed in the smaller bronchi, and then, by coughing, being loosened and pushed up a certain distance; while this is going on the mucous membrane continues to secrete mucus which forms the peripheral part around the central part, made up of irregular whirls and masses which, as they are formed, have been pushed up. When you get down into the smaller bronchi the cast is solid, and under the microscope you will notice a mucous or gelatinous matrix, and in it fine fibrils that run in various directions, sometimes interlacing. If that is stained by methods that demonstrate fibrin you find that a certain number of these threads, varying in different cases, will take the fibrin stain, the ordinary Weigert fibrin stain, which shows that some of the fibrils by staining reaction are fibrin. In other words, the matrix seems to be made up of mucin, it gives the staining reaction of mucin in which are fibrils, some of which may be fibrin.

There is an intestinal condition which in many ways is very similar to fibrinous bronchitis. This intestinal condition is

pretty common, but fibrinous bronchitis is pretty uncommon. That intestinal condition is what you are probably very familiar with as mucous colitis. It is a condition in which the patient passes casts of the intestinal tract, largely of the lower part of the colon, often tubular, and rather apt to be bile stained; otherwise they have the same general appearance as the casts that this patient coughs up. If you look at the intestinal casts under the microscope, you see they are made up of mucus, and in that mucus runs a certain number of fine threads or fibrillæ, and, if stained, you find that it is partly mucin in reaction and partly fibrin. Mixed in with the casts you find a certain number of cellular elements which in the intestinal casts are usually lymphocytes, red blood-cells, and a certain number of desquamated epithelial cells.

Mucous colitis is a condition which has just the same indefinite etiology as this so-called fibrinous or plastic bronchitis. In both conditions there is, in most cases, an element of neurosis; they are apt to occur in people of rather unstable neurotic make-up, people of the type with rather active reflexes, etc. The same thing you know is true of asthma. A great many of the asthmatics have this indefinite neurotic element.

In the case of fibrinous bronchitis usually you find mingled in with the casts, more on the surface than inside, varying kinds of bacteria, and cultures made from the sputum will show a flora, which, after all, is not very different from the flora that you get in various forms of chronic bronchitis and which you get in asthma. This particular patient's culture showed a hemolytic streptococcus and Streptococcus viridans, and from his sputum culture a vaccine has been made, and we have been using that in treating the case.

Probably what has happened in this patient is that he developed some form of bronchitis which is persistent in character, not very severe in nature, and along with that from time to time has occurred the formation of these casts, and his very violent spells of coughing have been at times when these casts were rather extensive and vigorous coughing is necessary to expel them. Running along through the whole thing has been per-

sistent bronchitis. You heard him coughing since he has been in here, a certain wheezing cough with a little rattle to it, just the kind of cough the asthmatic has between asthmatic attacks.

In this condition esosinophilic cells are a common finding in the sputum, free and attached to Curschmann spirals, and this man's sputum shows a considerable number of these eosinophilic cells, but no spirals. In the descriptions of the casts very often it is said that the terminal portion.of the cast is of the structure of a Curschmann spiral. In that picture passing around you see at the tip of the cast a very fine terminal structure which shows a tendency to be twisted on itself. The Curschmann spiral you know has a mucous matrix with one or two threads of more highly refractive material running in it, and the whole thing is twisted on itself, hence the name spiral; so very often these casts are described as showing Curschmann spirals at the tip of the branches.

It is not at all unlikely that there will be some relief from the condition following the use of the vaccine very much in the same way that we often see improvement in chronic bronchitis and asthma from a vaccine made of the predominating organism in the sputum. The therapeutic effect of adrenalin which is quite striking in this case is quite similar to the therapeutic effect of adrenalin in asthma, probably due to the same reason, namely, wheezing and difficulty in breathing is from bronchial spasm and that is relieved by adrenalin. In this particular case it may be of the nature of an ordinary asthmatic attack or simply may be a reflex spasm from the local irritation of the cast material which occupies the bronchus. If you have ever inspired a crumb you know the kind of a violent cough it will lead to, and when the coughing keeps up you are apt to go into this wheezing type of dyspnea. That is undoubtedly due to reflex spasm because ordinarily when one inspires a crumb it does not go very far down into the bronchial tree, it goes down a certain distance, but the dyspnea is largely a reflex affair.

It was described in this case that when he first came in the diaphragm apparently moved very little, that is, the excursion at the base of the lungs was very slight. We find that in various

kinds of spasmodic breathing, often very commonly in the cases of asthma, so that with the patient in front of the fluoroscope, when having the asthmatic attack, you can see that the lungs are blown up on account of the difficulty in expiration, and you can see that the diaphragm has a low position, and that with each inspiration the motion of the diaphragm is very slight. With the patient in front of the fluoroscope, if you will give him a subcutaneous injection of adrenalin, the dyspnea will be relieved, and the change that you can see take place is an increase in expiration, and an increase in the density of the lung, while the diaphragm has a more evident motion; gradually there is wider and wider excursions, until you get normal movement of the diaphragm, and from being a flattened diaphragm it becomes changed to a normal dome-shaped diaphragm at a higher level. That is the effect of the adrenalin. Very likely in this case the adrenalin has very much that therapeutic effect. The striking thing in this history has been the fact that adrenalin relieved the attacks of dyspnea promptly, but did not have much effect on the underlying condition. The dyspnea kept up just the same as far as recurring attacks were concerned, and it seemed to him as if he wheezed a little more throughout the next day after the attack was relieved. That is the same sort of thing that you find quite frequently in asthma.

Whether the injection of the vaccine with the use of the potassium iodid has made this striking change in the patient, or whether it is an effect of change in environment I do not think that you can say. It does not follow because of his relief immediately following a certain therapeutic measure that the therapeutic measure has produced that relief. The history of this condition is one of attacks lasting, as this one has, for several months, then gradually subsiding and leaving normal respiration. In some patients the attacks occur but once, and do not recur. In other cases they recur, such as in the negress whose history I gave you, who apparently every autumn for some time had been having these attacks. In other cases an irregular recurrence takes place over a number of years, so it may be that three months of attacks is about the length he is going to have, and the thera-

peutic measure really has not been effective. On the other hand, knowing of the results we have gotten in chronic bronchitis and asthma with sputum vaccine, that seems the rational form of therapeutics to carry out, and the response would seem presumptively one of cause and effect, a result of treatment. Of course, the idea of potassium iodid is to increase somewhat the bronchial secretion, really to have a larger amount of serous mixture, and probably dilute the mucus enough to prevent the formation of the casts, or anyhow to have more liquid and have the casts less closely applied to the mucus membrane and consequently more easily raised by coughing.

Apart from this condition the patient is normal. There is no evidence of any cardiac lesion, no evidence of any respiratory lesion beyond this, no evidence of any tuberculosis, and no evidence of any renal lesion. The blood-pressure is a little below normal rather than above normal, and between attacks he has a slight suggestion of chronic bronchitis, and with the attacks he has this dyspnea.

We have seen in this clinic a great many cases of cardiac disease with nocturnal dyspnea, but we do not find in those cases the type of sputum such as is being passed around, quite abundant sputum with casts in it. We have never seen the casts in any previous case. It is fair to say that they might be easily overlooked unless the sputum were examined macroscopically. There is an unfortunate tendency among students and house-officers to look at everything with the microscope and not look at it with the naked eye. It is like the proverb in the Bible of the mote and the beam, the very important beam is very often overlooked and the insignificant mote is carefully described, so I should not like to say we have not had any cases with casts in the sputum, but I can say that nobody has found any casts. Dr. Walker has been examining his asthmatics rather more carefully in regard to sputum, etc., and I think his figures are more reliable than the figures from our records, and he has not seen any of these casts.

A great deal can be learned from the sputum by doing two things which are relatively infrequently done, one is measuring

the sputum and knowing how much sputum a patient raises, whether it is increasing or decreasing. That ought to be done in all cases where there is chronic bronchitis, and especially bronchiectasis, because the variation when there are large cavities is a striking thing. The sputum is rarely measured, so the average student has very little idea as to whether the twenty-four-hour sputum should be 10 c.c. or 10 liters. You would be quite surprised often, I think, to find how much sputum a patient, who does not seem to cough very often or raise very much at a time, succeeds in bringing up in twenty-four hours. The other thing I have already referred to, and that is the macroscopic examination which tells you about bacteria to a certain extent from the smell and from the color of the sputum. Observation of the amount, color, and odor of sputum gives you a great deal of information. The odor is very important, and yet I doubt if anybody smelled that specimen as it went around to see whether it had an odor or not. Does anyone know what it smells like?

STUDENT: It has a certain aromatic smell.

DR. CHRISTIAN: What is that due to?

STUDENT: Probably medicine.

DR. CHRISTIAN: That medicine might have been put in the patient or put in the sputum. What does it smell like?

STUDENT: Like some coal-tar product.

DR. CHRISTIAN: Yes, it smells like some sort of thing that was put in there to keep it sweet and preserve it. It is certainly not a foul smell such as you often get from a case of bronchiectasis or lung abscess. If we have any sputa of that variety I will bring it in and let you smell it. It is an entirely different proposition.

You see from the odor of that sputum and the fact that there is very little odor to it, there is presumptive evidence that this patient has not any dilated bronchi in which the liquid material accumulates for a long time and is mixed with bacteria that causes putrefactive changes. That is what you get in bronchiectasis. This sputum, though abundant, is suggestive of the odor of sputum that comes from a condition not having much dilatation of the bronchi.

Examination by *x*-ray gives this picture, which probably represents more peribronchial thickening than dilatation of the bronchi. Of course from the *x*-ray picture you cannot tell whether a shadow running down, such as this, is due to thin-walled dilated bronchi or due to normal bronchi, as far as the bore of the tube is concerned, with thickened wall.

The important thing in this patient is the future as to whether he is going to have a recurrence of the condition or not and the best way to prevent that probability. I am inclined to think that a patient with this sort of history should live very much the out-of-door life of the case of tuberculosis that is well enough to be at work. That is, he ought to sleep with his windows wide open and get as much fresh air as possible and ought to be over-nourished. There is no indication for him to keep at rest if the paroxysms of dyspnea do not interfere with his work. I think it is decidedly advisable for him to have a course of vaccine treatment on the basis that there may be an underlying chronic bronchitis of bacterial origin, and the vaccine will possibly decrease that, and so decrease the occurrence of these paroxysms of dyspnea due to the accumulation of these casts. With that sort of general hygiene he may remain entirely free from these attacks the rest of his life.

One etiologic factor I referred to in giving his history, saying there was no exposure to irritating fumes. Some of the cases of acute fibrinous bronchitis have followed acrid fumes, such as ammonia fumes or the gas that comes from a fire, where the patient has inspired the smoke and gas in a burning building. Then there is a condition which is a terminal process following the inspiration of these irritating gases, which is spoken of as bronchiolitis fibrosa obliterans, where the gases have led to inflammation in the finer terminal bronchioles with subsequent connective tissue formation in the wall and obstruction of the fine terminal bronchi. That is an extremely rare condition, but it is well enough to keep it in mind in connection with this other condition which is relatively a rare one.

CLINIC OF DR. JOHN LOVETT MORSE

CHILDREN'S HOSPITAL

INFANTILE SCURVY

January 17, 1919.

THIS baby, who is nine months old, comes to the hospital from one of the larger cities of Canada. His father was told by several physicians there, including an eye specialist, that the baby's condition was incurable. His family physician told him that it was useless for him to take the baby to Boston, but, as the baby's mother would never be satisfied if he was not taken there, it would be best for him to go.

The parents are well. There are no other living children. One child died at twenty months of some disturbance of the digestion. There have been no miscarriages.

He was born at full term after a normal labor, was normal at birth, and weighed 5 pounds and 11 ounces. He was nursed for six months. He was then fed on pasteurized cow's milk, modified according to the formulæ given by the proprietors of Phillips' Milk of Magnesia. The present mixture is made up of the

Top 9 ounces of a quart of milk.
9 ounces of barley-water.
1 ounce of milk of magnesia.
1 ounce of milk sugar.

The milk is still pasteurized. This mixture contains about 5.50 per cent. of fat, 7 per cent. of milk sugar, 1.50 per cent. of protein, and 0.75 per cent. of starch. He has been taking five feedings of 8 ounces at four hour intervals. He has not vomited and has had one or two normal stools daily.

It has been rather difficult to get accurate data as to the

sequence of events, the mother, the aunt, the trained nurse, and the nursery-maid all giving different dates. As far as I can make out, however, it was noticed about six weeks ago that he cried when his diapers were changed and when he was given his bath. A little later he acted as if his back was tender, and recently his legs have been tender when they were touched. Little attention had been paid to the tenderness in the legs and back, however, because of the more marked symptoms in the eyes. Three weeks ago his left eye began to bulge, and within fifteen or twenty minutes was very prominent. A large ecchymosis appeared shortly afterward in the left upper lid. Ten days later the right eye also protruded, and an ecchymosis appeared in the right upper lid. He had a slight nosebleed at the time when the right eye swelled. A discoloration was noticed in the roof of the mouth at about the same time. The color of the urine has not been noted.

Physical Examination.—He is fairly developed and nourished, but markedly pale. The anterior fontanel is 3 cm. in diameter and level. The head is of good shape. Both eyes are, as you see, very prominent, so prominent that he cannot quite close the lids. The conjunctivæ are, however, not inflamed. There is no limitation of the motions of the eyeballs. The pupils are small, but react quickly to light. Both upper lids are stained bluish-brown and the lids are a little puffy. He sees well, and there is no trouble with his hearing. There are no teeth, and the gums are healthy. The tongue is clean. There is an ecchymosis about 3 cm. long and 2 cm. wide on the posterior part of the hard palate in the median line. There is a moderate rosary, but the chest is of good shape. The right border of the cardiac dulness extends 2½ cm. to the right, and the left 6½ cm. to the left of the median line. That is, the heart is slightly enlarged to the right. The action is regular. The rate is 160. There is a slight systolic murmur at the pulmonic area. The second sound at the pulmonic area is normal. There is also a slight systolic blow in both sides of the neck. The lungs are normal. The level of the abdomen is that of the thorax. Nothing abnormal can be detected in it. The lower border of

the liver is palpable, 1 cm. below the costal border in the nipple line. The spleen is not palpable. He uses his arms freely and, as you see, they are not tender. There is, however, some swelling over the lower portion of the diaphysis of the radius on each side. This swelling is deep down and is slightly tender. It is not, however, red or hot, and does not pit on pressure. When I prick his feet with a pin he draws his legs up, as you see, but shows by his actions that he would prefer not to move them and that it hurts him to move them. There is, nevertheless, no limitation to passive motions at any of the joints. There is a little swelling about the shaft of the right femur. This swelling is very tender, but is not red or hot, and does not pit on pressure. There is some swelling about the lower portions of the shafts of the tibiæ and fibulæ on both sides. These swellings are also tender, but not red or hot. They do not involve the epiphyses or the joints. The knee-jerks are equal and unusually lively. Kernig's sign is absent on both sides. There is no enlargement of the peripheral lymph-nodes. The rectal temperature is 102° F. The pulse is 160. The respiration is 60.

The urine is light red in color, turbid, acid in reaction, and of a specific gravity of 1022. It contains a very slight trace of albumin, but no sugar. There is 1 cm. of sediment in the centrifuge tube. This contains many red blood corpuscles and a few white corpuscles, but no other cells or casts.

The stool contains a slight excess of soap, but is otherwise normal. The guaiac test for occult blood is negative.

The hemoglobin by the Sahli test is 45 per cent. The red corpuscles number 3,500,000, and the white corpuscles, 18,000. The red corpuscles show some achromia and anisocytosis, but no polychromatophilia, poikilocytosis, or nucleated forms. The differential count of the white cells shows:

Mononuclears	60 per cent.
Polynuclear neutrophils	39 "
Eosinophils	1 "

Diagnosis.—I am sure that you have all already made the correct diagnosis, because I know that you are familiar with this

disease. You are all, I am sure, also congratulating yourselves upon being so much brighter than the physicians in the Canadian city. You must not feel too much pleased with yourselves, however, because ten years from now, when you are not in touch with large hospital clinics and when you have not seen one of these cases for a good many years, more than half of you will fail to recognize it. To one who is familiar with this disease there can be, of course, no differential diagnosis in this instance, because there is no other disease but scurvy which gives these characteristic symptoms and physical signs. The tenderness of the legs and back on handling and the peculiar situation and characteristics of the swellings about the bones of the extremities are absolutely pathognomonic. The blood in the urine, without casts or cells, is also characteristic of the hematuria of scurvy and inconsistent with acute nephritis. The sudden protrusion of the eyes could only be caused by a hemorrhage into the orbit. No new growth could have forced the eyes forward so quickly. Furthermore, if the protrusion of the eyeballs was due to an increase in the intracranial pressure, there would be also bulging of the anterior fontanel and evidences of cerebral irritation. The ecchymoses in the eyelids are also corroborative of this etiology, as is the bleeding from the nose at the time of the protrusion of the right eye. The ecchymosis in the roof of the mouth is also corroborative evidence of scurvy. There would be, in all probability, swelling and discoloration of the gums if there were any teeth. Further, but less important, evidences of scurvy are the anemia, the slight enlargement of the heart, the rapidity of the pulse and respiration, and the exaggeration of the knee-jerks. The murmur at the pulmonic area and the systolic blow in the neck are unquestionably functional in character and of no importance. The elevation of the temperature is not inconsistent with scurvy, although many of the text-books give the impression that uncomplicated scurvy is never accompanied by fever. I have, however, seen it very frequently.

The most interesting feature in this case is that the hemorrhages into the orbits have been so large, when the other signs of scurvy have been so mild. It is also interesting to note that

the signs of rickets are so slight, although the scurvy is so marked. The mixture being a perfectly rational one, the cause of the scurvy in this instance is almost certainly the long-continued use of the pasteurized milk.

Prognosis.—The prognosis is absolutely good with proper treatment. The temperature will drop to normal within a few days and the blood will also disappear from the urine. The tenderness in the back and extremities will probably all be gone within a week. The eyes will quickly begin to go back into their normal position, but it will probably be two or three weeks before they will be quite where they belong. It will take some time, of course, for the ecchymoses in the eyelids and in the roof of the mouth to disappear.

Treatment.—The treatment is very simple, namely, 2 tablespoonfuls of orange juice once daily. This should all be given at one time one hour before some feeding. It may be given plain or diluted with water. If the baby dislikes it, it may be sweetened with a little granulated sugar.

The baby will recover, even if the pasteurized milk is continued. There seems to be no reason for continuing it, however, because a clean and safe milk can be provided for the baby. It is apparently digesting very well the mixture which it is taking. It seems irrational, however, to be giving a baby so much magnesia, and the stool shows that the baby is not quite taking care of the fat in its food. We will give it a mixture containing 3 per cent. of fat, 7 per cent. of milk sugar, and 1.50 per cent. of protein, adding 2 grains of citrate of soda to each ounce of milk and cream in the mixture to take the place of the milk of magnesia in facilitating the digestion of the casein. We will give the baby five feedings of 8 ounces at four hour intervals.

DISCUSSION OF INFANTILE SCURVY

Symptomatology.—Using this case as a text, I now want to tell you something of this disease as it occurs in infancy. The earliest symptoms of infantile scurvy are pallor, edema of the eyelids, especially of the upper, loss of appetite, disturbance of the growth and nutrition, and cardiorespiratory disturbances.

There is nothing characteristic about these symptoms, which may occur, of course, in many other conditions, except, perhaps, the cardiac-respiratory disturbances. Even these may also be due to other causes. The occurrence of any of these symptoms should, nevertheless, always suggest the possibility that scurvy is developing. The only way to determine whether these symptoms are due to scurvy or to some other cause is by the therapeutic test. If they are due to scurvy, there will be an immediate improvement when orange juice is given. It is very surprising, unless one is familiar with it, to see how rapidly the appetite will improve when orange juice is added to the diet, and how a baby, whose weight has been stationary for weeks, will begin to gain at once when orange juice is taken.

After a time the characteristic symptoms of scurvy develop. These may be divided clinically into three rather definite types. Usually, for a time, at least, the symptoms are entirely of one type, but in the well-developed cases the symptoms of all types may be present together.

In the ordinary type the first symptoms noticed are tenderness of the legs and back when the baby is handled. This is usually first noticed when the diapers are changed or the baby is bathed. The tenderness becomes more marked and the baby uses its legs less and less, finally not moving them at all unless compelled. Tenderness and pain on motion may also appear in the arms. After a time swelling appears about the long bones of the legs or of the arms. This swelling is due to hemorrhage between the bone of the shaft and the periosteum, and is limited, therefore, to the diaphysis. The swelling is tender and tense, but is not hot or red, and does not fluctuate. It does not involve the epiphysis or the joint unless, as happens in some of the severer cases, the hemorrhage has broken through the periosteum and into the tissues about. This swelling occurs most often about the femur, but is not uncommon about the lower parts of the tibiæ and fibulæ. It occurs more often in the forearms than in the upper arms. It is almost always bilateral. The position of the child is most characteristic. It lies on its back with its thighs flexed on the body and rotated outward, and with the

knees flexed on the thighs, that is, in the position which produces the least muscular strain, and is, therefore, the most comfortable. This position is fairly well shown in Fig. 237.

The baby does not move its legs. This is not because it cannot move them, but because it hurts it to move them. The legs are, therefore, held rigidly and are not flaccid. Passive motions at the joints are not limited except in the most severe cases. The knee-jerks are normal or exaggerated, and there is no disturbance of the sensation.

In the second type, the first symptom is swelling and discoloration of the gums about the teeth. The swelling is soft and

FIG. 237.—Position of legs in scurvy.

spongy, and the color purple. It occurs most often about the incisor teeth. It usually appears first and is most marked about the upper teeth. It may be present behind the teeth and not in front. Swelling and discoloration of the gums almost never develops unless the teeth have erupted, but does sometimes, however, when no teeth are visible.

In the third type the first and, often for a considerable length of time, the only symptom is the presence of fresh blood in the urine. In these cases the urine is red and contains albumin, but shows no casts or renal elements. There are no urinary symptoms except the discoloration of the urine.

In addition to these symptoms, which, as I have said, may all be present at the same time in advanced cases, there are other symptoms which are important, but less characteristic. Hemorrhages are the most common of these symptoms. They may occur anywhere in the skin, making ecchymoses of various sizes. Hemorrhages may also take place into the orbits, as happened in the baby that we have just seen. Bleeding from the nose or into the conjunctivæ is not uncommon. Cerebral hemorrhages, hemorrhages into the joints and hemorrhages from the bowels are, however, most uncommon. The pallor, which has been mentioned among the early symptoms, is always a marked symptom in the well-developed cases. Edema is also a not uncommon symptom of scurvy. This may occur in the eyelids, as already mentioned, or in the face or dependent portions. In addition to this ordinary type of edema there is also a very firm type of edema which occurs about the bones, especially of the lower legs. This type of edema does not pit on pressure and may easily be mistaken for hemorrhage about the bones. It is often a very confusing condition, and sometimes, if its real nature is not recognized, obscures the diagnosis.

Fever, even as high as 104° or 105° F., is not at all uncommon in the acute cases, although many of the text-books state that fever does not occur in infantile scurvy. I am sure that it does, however, because I have so many times seen a high temperature in scurvy without anything to account for it except the scurvy, and have then seen this temperature drop to normal in a few days, with the subsidence of the symptoms of scurvy under suitable treatment.

Certain evidences of neuritis, such as exaggeration of the knee-jerks and edema of the optic disks, are present in some instances. Enlargement of the heart, especially to the right, is not infrequent. An increase in the rate of the pulse and the respiration, especially of the pulse, is also not uncommon. The baby that we have seen today shows many of these minor signs, that is, exaggeration of the knee-jerks, enlargement of the heart to the right, and increase in the rate of the pulse and respiration.

Pathology.—I do not wish to take up the pathology of scurvy in detail. There is anemia in all cases. This anemia is of the secondary type, and shows the peculiarities of anemia of this type in infancy. That is, there is a tendency toward the chlorotic type of blood, to leukocytosis, and in the severe cases to reversion to a younger type of blood. The hemorrhages about the bones and their characteristics I have already spoken of. The bones themselves show some rather characteristic changes. The bone-marrow loses its lymphoid character and is poor in

FIG. 238.—Small hemorrhage beneath periosteum.

cells. It contains relatively few blood-vessels. There is much calcified ground substance, but it is not converted into true bone. The cortex of the bone is thinner and more brittle than normal. The density of the bone is diminished at the epiphyseal line. Fractures and separation of the epiphyses occur, therefore, more readily. The periosteum of the long bones is thickened and congested. The hemorrhages occur as the result of this congestion. In many instances there are also hemorrhages in the bone-marrow. These radiographs (Figs. 238–240) show some of these changes.

It is presumable that there are certain changes in the nervous system because of the exaggeration of the reflexes and the changes which have been found in the optic disks. These changes have, however, never been carefully studied.

The cause of the hemorrhages is still somewhat doubtful. The blood shows a slight diminution in clotting power. This diminution is not due to a deficiency of calcium nor to an increase in the antithrombin. The blood-platelets are normal. The

FIG. 239.—Large hemorrhage beneath periosteum with slight displacement of epiphyses.

changes in the blood do not seem, therefore, sufficient to account for the hemorrhages. Hess believes, from the use of what he calls the "capillary resistance test," that the tendency to hemorrhage is due to changes in the vessel walls and not to the blood. In this test he used a blood-pressure band to determine at what point petechial hemorrhages occur into the skin. Normal infants withstood 90 degrees of pressure for three minutes, but the vessels of infants with scurvy gave way under this pressure. This test is, however, not specific for scurvy.

Etiology.—There is not time for us to go very deeply into the various theories as to the etiology of infantile scurvy. It is most common in the second six months of life, presumably because so many babies get breast milk during the first few months of life and because after they are a year old they begin to get a more general diet. Sex plays no part in its production. We know furthermore that it is not due to improper hygenic

FIG. 240.—Large hemorrhage beneath periosteum with considerable displacement of epiphyses.

surroundings, to congenital or inherited debility, to other diseases, whether or not of nutrition, or to inanition or starvation.

Experiments on animals show that it is not due to starvation or to the long-continued use of a single article of food. They suggest that it is due to the lack of some substance in the food which is destroyed by heating or drying.

Clinically, it occurs very rarely in the breast fed, but may develop on any or all kinds of food. The conclusions of the American Pediatric Society, after its investigation on infantile

scurvy in 1898, were that "the development of the disease follows in each case the continued employment of some diet unsuitable to the individual child" and that "the farther a food is removed in character from the natural food of the child, the more likely its use is to be followed by the development of scurvy." In spite of the vast amount of experimental work which has been done since this time, no more positive conclusions are really justifiable now than when these conclusions were made twenty years ago. In my own experience, the most constant of the factors which may have been of etiologic significance has been the continued heating of the food. Clinically, the disease is easily cured by the addition of certain things to the diet, such as fruit juices and potato. Furthermore, when scurvy develops in babies that are taking heated milk, recovery takes place when the heating of the milk is stopped. All of these things seem to show pretty definitely that infantile scurvy is not due to the presence of some abnormal element in the food, but to the lack of some essential element.

It seems reasonable to believe that the element lacking is one of the so-called "vitamins," and that scurvy is one of the "deficiency diseases," belonging in the class with beriberi, pellagra, and perhaps rickets. The vitamins are sensitive to heat. This fact would explain the effect of heating the milk in the causation of scurvy. When, however, it develops in babies that are taking raw milk, we have to assume that there is a deficiency in the vitamins. Such a deficiency is possible, because it has been found that the amount of the vitamins in milk varies with the food and the nutrition of the animal, whether it be cow or woman. This fact will explain the occasional development of scurvy in breast-fed infants. The lack of growth in length, the evidences of neuritis, the edema, and the cardio-respiratory disturbances are also suggestive of the other deficiency diseases, especially beriberi.

A great deal of experimental work as to the etiology of scurvy has been done in animals during the past three or four years. The results of these experiments are somewhat conflicting. The conclusions which have been drawn from many of them are

probably erroneous, in that the experimenters have failed to realize that conclusions cannot be drawn from one species of animals and applied to another species, or from animals and applied to infants. The theory, which gained considerable credence about two years ago, that scurvy is due to stagnation in the colon with the absorption of toxins or bacteria, has been apparently disproved by more recent experiments. So also has the idea that the action of orange juice and lemon juice in scurvy is simply laxative rather than specific.

Diagnosis.—The diagnosis of scurvy is impossible in the early stages before the development of the characteristic symptoms. It should, however, as I have already said, be suspected when babies become pale, have edema of the eyelids, lose their appetite, cease to gain in weight and length, or have a rapid pulse and respiration. When these symptoms occur and there is no other obvious cause for them scurvy should be suspected, and the treatment for scurvy instituted. If scurvy is the cause of the symptoms, they will be quickly relieved.

Anyone who is familiar with the type of scurvy, beginning with pain and later showing swelling of the extremities, will never mistake it for anything else. Those who are not familiar with this type, however, make various other diagnoses, such as rheumatism, periosteitis, osteomyelitis, sarcoma, hip disease, Pott's disease, injuries, infantile paralysis, and multiple neuritis. Such mistakes ought not to be made. Rheumatism does not occur in infancy. If it did, and there were any swellings, the swellings would be about the joints and not about the bones. There would be heat and there would be redness. Periosteitis and osteomyelitis are serious, acute diseases. Babies with them are manifestly seriously ill. The trouble is usually limited to one bone or one extremity. Tenderness is sharply localized. If there is swelling, it is usually red and hot. The temperature is usually higher and more irregular. There is a marked leukocytosis. Sarcoma occurs in only one bone of one extremity. The swelling is usually not tender and is usually very hard. There is usually no fever and there are no other signs of scurvy. Hip disease is unilateral, there is limitation of motion at the

hip, the tenderness is over the joint, and swelling, if it occurs, is about the joint. In Pott's disease there is rigidity of the spine and usually an angular deformity. Psoas contraction is usually present. There are no other evidences of scurvy. Injuries are usually unilateral, sharply localized, acute, and there are no other signs of scurvy. Infantile paralysis is an acute disease. The lack of use of the extremities is due to paralysis and not to pain. The extremities are flaccid and are not held rigidily. There is no swelling. The reflexes are diminished or absent. Multiple neuritis is not a disease of infancy. The loss of use is due to paralysis and not to pain. There is, therefore, flaccidity, not rigidity. There is no swelling. The reflexes are diminished or absent. Many other minor points might be given in the differential diagnosis between scurvy and these other conditions. The points, which I have given you, however, are sufficient to prevent mistakes.

The type with swollen gums is often mistaken for difficult dentition. This mistake should never be made. The swelling of the gums in scurvy almost invariably occurs about teeth which have erupted, or are in process of eruption. The swelling in difficult dentition occurs above a tooth which has not erupted. The swelling in scurvy is purple and soft. The swelling in difficult dentition is red and hard. The eruption of the tooth relieves the symptoms in difficult dentition. The eruption of a tooth in scurvy has no influence on the symptoms.

The type of scurvy beginning with hematuria is almost never recognized. It is mistaken for nephritis, or the hematuria is attributed to stone in the urinary passages, tuberculosis or sarcoma. Sarcoma of the kidney, it is true, is sometimes associated with hematuria. The hematuria is, however, slight and intermittent, not marked and continuous. There is, moreover, almost always a palpable tumor in the region of the kidney if there is hematuria. Tuberculosis of the kidney and bladder in infancy is a pathologic curiosity. Hematuria almost never occurs in it. The urine, if it shows anything abnormal, contains pus. Stone in the urinary passages is also most unusual, although irritation of the urinary passages from crystals with

hematuria as the result sometimes occurs. In such cases the crystals can be found in the urine. If there is any doubt, the administration of an antiscorbutic will clear up the diagnosis in a few days. When there is a nephritis, there are always casts and other renal elements in the urine. These are, however, almost never present in scurvy, or, if present, there are very few of them.

Prognosis.—The prognosis in infantile scurvy is good. Recovery is rapid and complete under proper treatment. Hematuria usually ceases inside of a week. Tenderness of the extremities is usually gone in a few days. The swelling, however, lasts much longer, because time is required for the absorption of the blood. Improvement in the swelling in the gums is manifest within forty-eight hours and the swelling is usually all gone inside of a week. Even in cases where there has been a separation of the epiphyses, the epiphyses almost invariably come back into place. It is only in the rarest instances that any permanent deformity of the extremities remains. I have myself seen only one case in which this occurred. In this instance the case had been neglected for a long time and there had been so much ossification of the elevated periosteum that it could not fall back into place.

Treatment.—The first element in treatment is the prevention of the disease. Knowing, as we do, that scurvy develops more frequently in babies that are taking foods made with heated milk or foods prepared without milk, it is always wise to give such babies orange juice or lemon juice to prevent the development of the disease. My experience has taught me that it is necessary to give at least 1 tablespoonful of orange juice daily in order to certainly prevent its development. Hess has suggested the use of potato water, which he has found effective in the prevention of scurvy, in place of the usual cereal diluents, because it is so much cheaper than the fruit juices. He has also suggested the use of the juice of the peel of the orange as an antiscorbutic, as it is both effective and cheap.

When scurvy has developed, it can usually be cured by changing the food. It can almost always be cured by stopping

the heating of the milk, if it is being heated. Recovery is slow when nothing is done but to change the food or to stop the heating of the milk. It is much wiser, therefore, to give an antiscorbutic at the same time, in order that the recovery may be more rapid. Recovery will take place, moreover, when an antiscorbutic is given, even if the food, which was the cause of the scurvy, is continued. The most available antiscorbutic is orange juice. In order to certainly get satisfactory results 2 tablespoonfuls of orange juice should be given daily. Less may not be sufficient; more is unnecessary. It is best to give the orange juice all at one time, one hour before some feeding. It is advisable to give it at this time in order that the stomach may be empty and that the digestion of the milk may not be disturbed. Orange juice may be given plain or diluted with water. There is no objection to sweetening it with a little cane sugar, if it seems desirable. The juice of the orange peel is equally as effective and can be used instead of the juice of the orange. Strange as it may seem, boiling the fruit juices does not destroy their antiscorbutic action.

Boiled and mashed potato also has a definite antiscorbutic action, but potato flour has none. Beef juice has a slightly antiscorbutic action, as have wheat middlings. Their antiscorbutic action is, however, not to be compared with that of the fruit juices and potato. Yeast, cod liver-oil, and olive oil have no antiscorbutic action.

There are no drugs which have any effect whatever in the prevention or cure of infantile scurvy.

CLINIC OF DR. WILLIAM P. GRAVES

FREE HOSPITAL FOR WOMEN

CANCER OF THE UTERINE BODY AS A BORDERLINE CASE IN GYNECOLOGY

HAVING been asked to give a clinical lecture on the subject of borderline cases in gynecology, I am confronted with a certain doubt as to what the expression borderline case really signifies. In fact, I do not for the moment recall having heard it used specifically in gynecologic classification. To my mind the term "borderline case" implies an indecision as to the exact status of a given case in its relation to two specialized branches of medicine, and this condition of doubt may refer to matters either of treatment or of diagnosis, or of both. In my medical school days a borderline case suggested a malady to which the general medical man and the general surgeon both laid claim. In those days the most conspicuous example of such a case was chronic appendicitis. Our eminent professor of clinical medicine was emphatic in his assertion that chronic appendicitis is essentially a medical disease, and should be treated as such, while our surgical instructors, with equal vehemence, held to the view that the chronic appendix should be removed. Viewed from this standpoint cases on the borderline between medicine and surgery are still numerous. If we look for borderline cases in gynecology, we shall find ourselves in a clinical field much too extensive to be treated in detail by a single lecture, inasmuch as cases will be found on a considerable number of different borderlines. For example, one who is interested in orthopedics would be surprised to see in a large gynecologic clinic how many patients apply for treatment on the supposition that they have pelvic disease, whereas, in reality, they are suffering from some bone

or muscle lesion. Again, the boundary lines of gynecology and neurology intersect very frequently, and it is a well-known fact that neurologic clinics abound with patients who need gynecologic treatment, and conversely. In like manner, it will be found that the broad science of gynecology has lines of attachment to practically every specialized branch of medicine, no matter how exclusive, and that at each point of contact there are cases which may properly be designated as "borderline" in the varied significance of the term. As far as general intersest goes, the most practical treatment of my subject would be to discuss those cases that may be said to lie in the borderland of practice between the general practitioner and the gynecologic specialist.

This of itself constitutes a wide range of subjects, but I shall confine myself to a single one which will help to illustrate certain points concerning which there is at present insufficient co-ordination between physician and specialist.

The patient before you is one whom I first saw professionally a little over two years ago. Though seventy-five years of age she was at that time a strong, rugged woman. She had had the menopause at about fifty, but had since then noted an occasional slight show of blood to which she paid little attention. About a year before her visit she had begun to have what she thought was incontinence of urine. She consulted her physician, who made light of the trouble, and without examining the patient prescribed something to allay bladder irritation. The watery discharge continued, however, and as it was rather offensive and was occasionally streaked with blood the suspicion had been aroused in the patient's mind as to whether the discharge really issued from her bladder.

Pelvic examination revealed a large, fixed, tender uterus which bled from the effects of palpation. The cervix appeared and felt entirely normal. There was a watery discharge from the uterine canal, the urine was normal. At the operation it was found that the uterine body was completely invested with a cancerous process which involved both tubes and one ovary. Notwithstanding the extensiveness of the disease throughout

the pelvic organs, *there was no involvement of the regional lymph-glands.*

Please note carefully the points in the pathology of the case, for they are of much significance in the matter of the clinical course of this disease. A complete hysterectomy was performed. The patient made a good recovery and remained in excellent health until about six months ago, or for about a year and a half after the operation.

She comes now for examination for recurring symptoms. Will you examine the patient, Mr. Meigs, and tell us what you find.

MR. MEIGS: I find an indurated area in the posterior wall of the vagina, high up, through which there seems to be some sort of an opening into the rectum. I think there are feces in the vagina. My diagnosis is recurrence of cancer in the vagina, with fecal fistula.

DR. GRAVES: Do you note anything special with regard to the vaginal wall?

MR. MEIGS: It seems unusually tough and leathery, and the whole vagina is curiously shrunken.

DR. GRAVES: Does the presence of a rectovaginal fistula in a comparatively early and well-localized recurrent cancer in the vault of the vagina suggest anything to you?

MR. MEIGS: It seems rather unusual. Perhaps you have been using radium in this case.

DR. GRAVES: You are quite right, Mr. Meigs, radium was given as a prophylactic several times after the operation. The tough shrunken nature of the vagina is characteristic of the result of radiation on normal tissues. The fistula is also undoubtedly the result of a too enthusiastic radium treatment. I have seen a number of postoperative cases like this, in some of which I have applied the radium myself, and in some of which it has been given by others, where fistulas have resulted. This experience has led me to discontinue the practice of using radium as a prophylactic after operation for uterine cancer. I doubt very much if it has any influence in preventing recurrence of the disease, while it causes great risk, even when carefully screened,

of producing vesical or rectal fistulas long after the time of its application, even if the disease does not recur. What is the prognosis in this case?

MR. MEIGS: Absolutely bad, sir.

DR. GRAVES: Could the patient have been saved with an early diagnosis?

MR. MEIGS: Very easily, sir. I have often heard you say that cancer of the body of the uterus is a very benign form of cancer, and that if it is taken anywhere near in time a permanent cure by operation can be practically guaranteed.

DR. GRAVES: Perhaps that is putting my remarks on the subject rather strongly, but nevertheless it is not far from the truth. Now, gentlemen, the history of this patient presents several very important points, which I hope you will remember when you get into general practice and begin to see gynecologic cases.

In the first place, a strong, healthy woman, notwithstanding her advanced age, had noticed for a considerable period of time an occasional very slight showing of blood which was so insignificant that it caused her no alarm. Supposing, Mr. VanStone, that such a patient should consult you, what conditions would you think of, excluding for the moment the possibility of cancer?

MR. VANSTONE: Well, I should think first of a cervical polyp.

DR. GRAVES: That's right, and why?

MR. VANSTONE: Mucous polyps of the cervix are apt to make their appearance after the menopause, and cause intermittent bleeding, quite similar to that of uterine cancer.

DR. GRAVES: Does the presence of a cervical polyp exclude a consideration of cancer of the body of the uterus?

MR. VANSTONE: I should hardly think so.

DR. GRAVES: No, it does not. On the contrary, the presence of a polyp suggests a tendency to activity on the part of the mucous membrane which makes it all the more imperative to investigate the true condition of the uterine canal. What else would you think of in this case?

MR. VANSTONE: Senile atrophy of the vagina will sometimes cause slight bleeding.

DR. GRAVES: That is right.

The epithelium of the mucous membrane of the vagina and vaginal portion of the cervix becomes thin and delicate in old age, and is very susceptible to superficial infections or chemical irritation from abnormal secretions higher up in the genital canal. The vaginal epithelium on account of its thinness readily desquamates under these conditions, leaving small denuded areas that are apt to bleed slightly or to cause a pinkish discharge. The symptoms are exactly like those of an early carcinoma of the uterine body. Now, Mr. Denney, supposing an elderly woman should consult you, complaining of slight hemorrhages from the vagina, and supposing you had examined her and found denuded bleeding patches on the vaginal membrane, would you be satisfied with making a diagnosis of senile vaginitis?

MR. DENNEY: No, sir.

DR. GRAVES: And why?

MR. DENNEY: Because cancer of the body of the uterus appears most commonly after senile atrophy of the genitals sets in, so that a bleeding vaginitis and a cancer of the body might exist at the same time.

DR. GRAVES: Yes, and what is more, the altered secretions from a cancer of the body of the uterus might of themselves set up a senile vaginitis. Here again, as in the case of the cervical polyp, a comparatively innocent lesion which could readily account for the bleeding symptoms should not satisfy the examiner and deter him from taking radical steps for a complete diagnosis.

In addition to cervical polyps and senile vaginitis there are, of course, other non-malignant causes of uterine bleeding after the menopause, such as sloughing fibroids, atrophic atresia of the cervix, myomatous polyps, etc., but these latter are less common and their diagnosis more obvious.

We will now take up the next important point of interest in the case before us. Mr. Thorndike, what symptom or sign of grave significance do you note in this patient's history besides the slight bleeding which we have been discussing?

MR. THORNDIKE: The watery discharge which she and her doctor took to be the result of urinary incontinence.

DR. GRAVES: What can you say as to urinary incontinence occurring suddenly in a healthy woman of seventy-five?

MR. THORNDIKE: I should wish to know whether the incontinence was functional or organic.

DR. GRAVES: Why?

MR. THORNDIKE: Because functional incontinence is usually due to a relaxation of the sphincter muscle of the neck of the bladder. I think it possible that a woman who has borne children might develop incontinence in advancing age. I should think it might also appear as a result of some urethral irritation like that of a caruncle or even from a colon infection of the urinary tract, which I understand is common in elderly people.

DR. GRAVES: As this patient described her symptom to me the watery discharge came away without her knowing it, at any time of day or night, so that she was obliged to wear a napkin continuously. What would you say to that?

MR. THORNDIKE: Under those circumstances the discharge if urinary must have represented an organic incontinence. I mean to say the urine must have escaped through a fistulous opening from the bladder into the vagina or urethra. I don't see how such an opening could suddenly be established in an elderly woman previously healthy. It might, of course, happen in an advanced stage of cancer, especially if radium had been used in her treatment. But there is nothing in this case to indicate that any cancerous process even if present was in more than an early stage when she first sought medical help. I believe there are certain lesions of the central nervous system which produce a complete organic incontinence, but I presume that these may be excluded in this case.

DR. GRAVES: What would be your conclusions?

MR. THORNDIKE: I should conclude that the watery discharge was not urinary at all, but that it probably issued from some disease of the genital tract.

DR. GRAVES: Very good. Then you would not, as this patient's doctor did, tell the patient without examining her that

the symptom of which she complained was incidental to her age and that it need cause no alarm.

MR. THORNDIKE: No, sir.

DR. GRAVES: On the contrary, the appearance of a watery discharge from the vagina should have served as a danger signal of the gravest nature, for it is perhaps more than any other one sign pathognomonic of uterine cancer. It is perhaps somewhat more constantly characteristic of cancer of the cervix than it is of cancer of the body, nevertheless, when it does make its appearance it usually means cancer somewhere in the genital canal. Occasionally a sloughing myomatous polyp will produce the same serous, watery, usually foul-smelling discharge seen in cancer. It always means danger. It represents an exudation of serum through the capillary wall of the numerous new vessels forming in the cancerous growth, and is frequently so profuse, thin, and colorless as to suggest to the patient that there is a leak in her bladder.

This patient whom we are considering did not receive the benefit of a pelvic examination when she first applied for medical assistance. Assume, Mr. Atwood, that she had consulted you when she first noticed the watery discharge, and gave you at the same time the history of having occasionally seen a little show of blood. Assume also that you very properly made a careful pelvic examination and found the vaginal mucous membrane perfectly smooth, the cervix small, atrophic and not indurated, and the uterine body small, movable, and normal in contour. What would have been your conclusions and what advice would you have given the patient? I shall expect you to answer in detail, as you have helped me operate on a number of these cases, so that you may give the rest of the class the fruits of your experience.

MR. ATWOOD: It is possible either for cancer of the cervix or cancer of the body to exist without the possibility of detection either by the palpating finger or by inspection of the vagina through a speculum. Cancer of the cervix nearly always has a suspicious feel to the examining finger, but sometimes the inverting type of growths may become quite far advanced be-

fore being palpable. I should not therefore jump at the conclusion that the patient did not have cancer of the uterus simply because I could not feel it or see it. In order to make more sure of the cervix, I should insert a speculum in the vagina and pass a small curet into the cervical canal. If cancer is present the curet will nearly always bring away a crumbly bit of tissue, with considerable bleeding. If I got a piece of tissue like that I should save it for microscopic examination. If I found no evidence of cervical cancer, I should tell the patient that my office examination was necessarily not complete, and that for final diagnosis it would be necessary for her to have the womb scraped under an anesthetic in order to make a microscopic examination of the lining membrane of the womb. If the patient objected or wished to put it off until some later date, I should urge the necessity of operation.

DR. GRAVES: And if she still persisted in her objections I should deliberately tell her I wished to find out definitely whether or not she had a *cancer*. The use of that word will always bring the patient to her senses. Go on, Mr. Atwood.

MR. ATWOOD: The next step would be to take the patient to a hospital, and, under ether, to curet the uterus, for the purpose of examining the curetings by microscope.

DR. GRAVES: Yes, and it is important to save the curetings for examination even if they are very small and do not suggest cancer by their gross appearance. A beginning cancer of the body in an atrophied mucous membrane may make a very insignificant showing in the curetings.

We will now return to the case under consideration, and you will recall that as a result of neglect the disease was found at operation to be very advanced and to have involved the uterine wall, the tubes, and the ovaries, but that there was no involvement of the regional lymph-glands. The local pathology of this case is very instructive in that it illustrates well the typical course that cancer of the uterine body takes. It always has its origin, as you know, in the endometrium, that is to say, in that part of the uterine mucous membrane which is above the internal os. At first the disease is localized, forming a wart-like ex-

cresence projecting into the uterine canal. The little papillary filaments of which the small growth is composed break off easily and cause a slight show of blood. This is a fortunate circumstance, for it gives the patient a warning signal early in the disease. The warning is especially apt to be heeded by the patient since cancer of the uterine body is more common after the menopause when the appearance of abnormal bleeding is more likely to cause apprehension. In this respect, as in every other, cancer of the fundus is less treacherous than cancer of the cervix, which may progress to a hopeless stage without a sign or symptom to indicate its presence.

Cancer of the body of the uterus progresses rather slowly. The disease spreads out over the mucous membrane and dips down into the muscular wall of the uterus. It has very little tendency to metastasize. Curiously enough, metastases when they do occur appear first in the ovaries, and this is a fortunate circumstance, for it tends to confine the disease to a tissue area which can easily be eradicated by operation.

Involvement of the tubes is by direct extension late in the disease. Invasion of the parametrium and of the cervix takes place only in very advanced stages.

Thus you will see, gentlemen, that cancer of the body of the uterus is from the standpoint of surgery a very favorable disease, in that it gives early warning, grows slowly, metastasizes late, and occurs in a group of organs which are easily removed. The percentage of permanent operative cures is very high, higher probably than in any other form of deep cancer. And yet it is extraordinary how many women who have this entirely curable disease will allow themselves, or be allowed by their physicians, to drift into a stage of inoperability.

Cancer of the body of the uterus, although most commonly a disease of the postclimacterium, is by no means confined to that period of life. It is frequently seen in the forties, and occasionally, though rarely, before. It is also often associated with fibroids, especially with those tumors that have attained a considerable size. The clinical diagnosis of cancer of the uterine body before or during the climacteric is less simple than

after the menses have ceased, as its symptoms are easily confounded with those of several other diseases which are apt to make their appearance in the fifth decade. A full discussion of cancer of the body at that period would entail a review of the whole subject of the menorrhagias and metrorrhagias of the menopause, which is one of the most important in gynecology, and one to which we have time in this lecture to make only a brief reference.

Cancer of the uterine body occurring near the menopause may, according to our special definition of the term, be justly classified with borderline cases for the reason that the abnormal bleeding that it causes is so frequently regarded by the general practitioner as a physiologic incident of the change of life, and therefore treated medically.

One reason why the diagnosis of cancer of the body at this period is so frequently missed by the physician is that it may give no palpable evidence of its presence in the uterus until the disease is considerably advanced. Cancer of the cervix usually causes at an early stage a suspicious hardness of the cervical lips which those physicians who take the trouble to examine their patients are, I am glad to say, learning to detect with increasing accuracy. Fibroids of the uterus are usually easy of diagnosis, especially as many of them can be readily palpated through the abdominal wall. In cancer of the body, however, the cervix is normal, and the size, consistency, and contour of the fundus may for a considerable time suggest no anatomic abnormality. In the absence of complicating fibroids, the clinical course, and findings from pelvic examination may exactly simulate those associated with the comparatively harmless condition of uterine insufficiency, which, as you know, is an extremely common affection of the menopause period. As most cases of uterine insufficiency will if the patient waits long enough become cured spontaneously, an erroneous impression has been created among the laity and among some of the profession that abnormally profuse bleeding is a physiologic phase of the menopause. This is a dangerous doctrine and has led to the death of many hundreds of women whose lives might have been saved by timely surgical interference.

I cannot be too emphatic in impressing on you that the normal menopause takes place by a sudden or gradual cessation of the menstrual flow. If at the climacteric age the flow is increased in quantity or in frequency or by a prolongation of the menstrual period, or by intermenstrual hemorrhages, the change must be regarded as abnormal, and immediate steps must be taken to insure an accurate diagnosis. In the absence of some obvious lesion, this can *only* be done by an examination under an anesthetic, with a removal by the curet of sufficient endometrium for microscopic section.

If this rule is rigidly carried out with every case of abnormal menopause bleeding it will be found that the majority of cases without palpable cause for bleeding will turn out to be uterine insufficiency. Nevertheless the number of cases of cancer in the whole number examined will be surprisingly large.

Subjecting a patient to the annoyance of a hospital ether examination and finding only a uterine insufficiency is not a matter to be in any way deplored. The ability to tell a woman who has been bleeding that she positively has no cancer not only gives immense mental comfort to the patient, but relieves the attending physician of a grave responsibility.

Moreover, uterine insufficiency, whether it be from a functional ovarian disturbance or from chronic tissue changes in the endometrium, is itself a disease which may have very serious effects on the patient's general constitution and especially on her nervous organization. If at the time of excluding the presence of cancer the abnormal bleeding may also be checked or the menstrual function completely stopped according as circumstances demand, the operation is still more desirable. Fortunately, we have in radium an almost specific cure for the hemorrhages of uterine insufficiency, and though at the present time it is available only in the larger medical centers, it will not be long before hospitals everywhere will be equipped with sufficient quantities for the treatment of this common and depleting affection.

In concluding the lecture I may sum up the points that I wish you to remember as follows:

1. Cancer of the body of the uterus may be classed as a borderline case because of the frequency with which it is treated medically by the general practitioner.

2. The slightest show of blood after the menopause should demand an immediate curetment of the uterus for microscopic diagnosis of the endometrium even if the bimanual examination reveals nothing abnormal to the touch.

3. Cancer of the uterus, both of the fundus and cervix, often causes a watery discharge which simulates urine. A watery discharge from the vagina is, therefore, a signal of danger.

4. Cancer of the uterine body grows slowly and is operable long after its initial symptoms.

5. Cancer of the uterine body is the most favorable for operation of all deep cancers because of its slow growth, late metastasis, and long confinement to a group of organs that can easily be removed.

6. Cancer of the uterine body may occur in the menopause decade, and simulates in its symptoms especially uterine insufficiency.

7. The menopause is normally characterized by a lessened flow of blood. Increase of blood at that time is an important danger signal.

8. Increased flow near the menopause should always be investigated by microscopic examination of the cureted endometrium, even if digital examination reveals no anatomic abnormality.

CLINIC OF DR. CHARLES J. WHITE

HARVARD MEDICAL SCHOOL

SOME COMMON ERRORS IN DERMATOLOGIC DIAGNOSIS AND TREATMENT

Errors of Omission and Commission in the Diagnosis and Treatment of the Commoner Diseases of the Skin. References to Certain Methods of Treatment which Do Not Figure in the Text-books.

As the years pass in my public and private consultation practice I am struck more and more by certain definite errors of omission and commission on the part of the general practitioner in his diagnosis and treatment of certain skin diseases. No great blame should be attached to such errors, for nowadays no one man who may be called upon to treat every conceivable human ailment can possibly know all that is known by those who give their entire time to one division of medical knowledge. What follows, therefore, is not to be taken as a personal criticism by any member of this audience, but should be received in the spirit in which it is given, *i. e.*, the crystallization of the thoughts of one who has witnessed these errors and seeks a method of preventing their future recurrence. What follows, then, will be a résumé of the commoner errors in diagnosis and references to certain methods of treatment which may not figure conspicuously in the text-books, but have proved of distinct worth in my hands.

ERYTHEMA MULTIFORME

There are individuals who are bothered with frequent recurrences of this disfiguring and often incapacitating disease which previous treatment has not cured. In such cases let me

advise you to try one or the other of the following methods internally, and remember that the drugs should be administered not only through the immediate attack but also for several months, even five or six, afterward, with one week's intermission out of every four. The first drug is hexamethylenamin in 5-grain doses after each meal in a full glass of hot water. If this fails after a thorough trial, then substitute calcium lactate to the amount of 10 grains in a half-glass of warm water after meals. To gain the full benefit of this latter drug under all circumstances the patient should eschew all acid foods, *i. e.*, raw fruits in any form, all types of pickles, tomato, cucumber, and vinegar, and should make a point of eating the lime-rich foods, such as milk, eggs, and spinach. Externally "white wash,"

℞. Acid. carbol..... 2
Calamin..... 4
Zinc. oxid..... 8
Aq. calcis.....q. s. ad. 250

always gives rapid relief to the burning and itching, and quickly dries up the ever-present edematous lesions. This wash should be sopped on (never bandaged) every hour until the lesions are stiff and dry, and when the skin seems about to crack from stiffness "Lassar's paste,"

℞. Acid. salicyl..... 0.65
Zinc. oxid..... 2
Amyl. maidis, vaselin.....aa 15

should be applied night and morning under one layer of old cotton or linen.

URTICARIA

The simple attack of "hives" following, in susceptible persons, the ingestion of certain well-recognized foods is usually easily relieved by a good saline purge, moderate fasting for a few days, and the application of any of the common antipruritics. There is, however, a decided proportion of these "sensitized" individuals who find their way, sooner or later, in despera-

tion to the dermatologist because endless previous medication has not relieved them from their intolerable itching and its resulting insomnia. These patients are usually women, and practically always of the "nervous," high-strung type, probably with a superfunctioning thyroid at the bottom of their troubles.

First of all, if possible, one must take these patients out of their present environment and occupations; rest should be the foundation of their cure. It is an accepted fact that a change of scene, of people, of food, of family surroundings is perhaps our best "tonic." If possible, arrange for such a change. For a few days starve these people on a diet of bread, butter, rice, and water; and then cautiously add simple roasted or broiled chicken or beef, baked or boiled potato, or macaroni, string beans, peas or spinach, and simple farinaceous puddings plus milk or freshly brewed tea. Great stress should be laid on the avoidance of all acid foods (see under Erythema Multiforme) and of coffee. Calcium lactate again is our standby internally. Every night, just before going to bed, these patients should take a starch or a soda bath. This means a warm bathroom; a pound of cornstarch or of cooking soda to the tub of water; temperature of water 95° F.; quiet immersion without soap for five minutes; drying by patting and not rubbing with a soft Turkish towel. Neither silk nor wool should be worn next the skin. Before the patient enters the bed some one should warm the sheets by passing a hot-water bottle between them. Cold and heat bring on paroxysms of wheals, and scratching and both extremes of temperature should be avoided. Externally an ointment of menthol,

℞. Menthol..... 1
Zinc. oxid..... 2
Vaselin..... 30

seems to be the most successful application.

If these combined methods fail, we can have recourse to the *x*-rays in analgesic doses, but this maneuver must never be undertaken by anybody but an expert. Such a combination of procedures practically never fails if co-operation has been sincere.

It may be necessary, however, in certain extreme cases to resort to skin tests to determine what is the noxious element present. Let me illustrate this point. Recently a young man was unsuccessfully treated according to the above methods, except that *x*-ray exposures were impossible because of his distant domicile in a small town. It was noted that his worst recurrences invariably followed an evening at the theater or the movies. Skin tests showed an intense susceptibility to rice, and it was evident that the exposure came through inhalation of rice powder from the skins of his "fair" neighbors in the hot, ill-ventilated auditoriums he frequented.

DERMATITIS EXFOLIATIVA

When confronted with this essentially scaling disease it is only natural for the practitioner to combat this condition in the customary way, that is, by the application of ointments. Dermatitis exfoliativa is a serious disease, and up to a certain date the skin records of the Massachusetts General Hospital showed that 50 per cent. of its patients afflicted with this disease had died under treatment. Then we began the so-called "dry treatment," and from that moment until the present day it is a fact that no patient with dermatitis exfoliativa, who has been treated *completely* by this method, has been lost. Nevertheless, the convalescence is commonly desperately tedious, the cure running into months.

The "dry method" consists in the superabundant application of borated talcum powder and imperatively nothing else. The patient should be placed in a room devoid of all carpets, rugs, hangings, pictures, and furniture—nothing save the bed is left. The hair should be cut close and the powder should be dusted on whenever in the twenty-four hours a single fraction of an inch of skin becomes visible. The patient should, in fact, lie in a bath of powder. Loose plugs of absorbent cotton should be placed in each nostril and there should be demulcent drinks for the dry throat and 10 per cent. argyrol solution for the eyes, because at times the facial orifices become painfully irritated by

the particles of powder floating in the atmosphere. All bathing is indefinitely suspended.

The first week or two of this intensive dry treatment may be, and usually is, a period of agony, for great cracks and crevices develop here and there over the body, and the patient and his family beg and plead for "just a little ointment" to soften the overlying crusts formed by the resultant oozing of serum. The physician must be as hard as adamant against these pleadings. This phase must be gone through with, and the sooner it is over, the better for all concerned, for afterward it never recurs, granting that there has been no digression, even by a hair's breadth, from the hard-and-fast line of the "dry treatment." If pus forms under the crusts, they must be forcibly lifted or cut away and more powder applied. It may be proper here to apply for fifteen minutes night and morning "black wash":

℞. Hydrarg. chlorid. mit..... 2
Aq. calcis..... 250

The comfort of the patient can at all times be enhanced by the use of an air-mattress and cradles to keep the clothes from the tender skin. Sterilization of all bed and body linen is obligatory, and a constant and steady supply should be on hand. The patient should be given an easily digested, light, but supportive diet, and possible intercurrent symptoms should be treated at once. There should always be plenty of fresh air, day and night, and suitable and cheerful companionship, if possible, should be provided. Let me repeat that the cure is discouragingly long drawn out; more than once we are tempted to try some other method, but let us always bear in mind that death has, as yet, never claimed any patient of ours who has been treated absolutely conscientiously by this method.

LICHEN PLANUS

The unwary physician has often been trapped into serious errors by this disease, either through ignorance or carelessness. The chief pitfall is the differential diagnosis between this disease and syphilis.

A papular syphilis may itch, and, in course of time, by the friction of scratching or of the clothes and by the natural subsidence of the lesions, the tops of the papules may become flat and glistening, and the sides may lose their original rotundity and the color may become tinged with blue, and then we are confronted, to a greater or lesser extent, with the subjective and objective symptoms of lichen planus. Let us all, however, remember the existence of this disease, and then with our modern means of diagnosis we can practically always prove the existence or the absence of dreaded syphilis.

There is, however, a form of lichen planus which can trap even the expert unless he be "on his toes," or unless he be a thorough routinist and has submitted his patient to the "dark-field" lens and the Wassermann test. This variety is known as lichen planus annularis, and curiously enough it has a decided predilection for the glans penis or its shaft or the scrotum or the lower abdomen. Lichen planus annularis and an annular syphilid require diligent circumspection and diagnostic acumen to differentiate between. History and modern laboratory tests will, of course, settle the question, but let us not forget the possibility of such a delicate clinical confrontation.

LICHEN SIMPLEX CHRONICUS (LICHEN CIRCUMSCRIPTUS)

These terms signify a definitely circumscribed, thickly infiltrated, dark red, minutely scaling, painfully itchy plaque somewhere on the trunk or the extremities, or at the base of the skull of a "nervous" individual—usually a woman and one approaching or in middle age. Endless drugs in various forms have been advocated and applied in this dermatosis, but despite these many previous failures, two methods of treatment have proved themselves trustworthy and successful.

If the attending physician is a man experienced in the cutaneous therapeutic use of x-rays he can soon bring about the relief so ardently desired by the long-suffering patient. If the physician cannot honestly qualify as such an expert, he should immediately send his patient to one who can. If this sacrifice on the physician's part is not possible, and very often, through

lack of time or money on the patient's part, or through distance, the transfer is not possible, then let him have recourse to crude coal-tar, and very likely the desired cure may be just as successfully and rapidly attained.

Crude coal-tar is one of the first products of the burning of bituminous coal—a black, tarry-smelling, viscous semisolid or liquid. One can now obtain it through the Eastern Drug Company. This substance, first introduced into dermatologic practice by Brocq, of Paris, has proved one of the great therapeutic "finds" of recent years. For use in the hair it should be combined with vaselin and applied between the hairs by means of a fine swab; for use on a dry surface it should be mixed with lanolin; and for applications to a moist condition it should be incorporated in a paste. It should perhaps be tried first in a 5 per cent. strength, and should preferably be diluted with an equal amount of zinc oxid. It is, unfortunately, a dirty, esthetically unacceptable medicament, and it smells; but its wonderfully successful therapeutic properties far outweigh its outward disadvantages. It naturally stains linen badly, but thorough rubbing with lard and subsequent washing in hot water and soap will usually remove all traces of discoloration. Before each morning and evening cutaneous application the old remaining portions should be removed with oil of sweet almonds—water should never be applied. During the use of crude coal-tar positively no other drugs should ever be employed locally; all hair at the site of applications should be kept closely cut; if pustules appear the inunctions should cease and not be resumed until all pus has disappeared; and there should be no bandaging over the affected area. Long experience has taught the necessity of these hard-and-fast rules.

PSORIASIS

From a diagnostic point of view we should not forget that in the macular, delicately scaling types of psoriasis the disease may closely resemble eczema psoriasiforme and pityriasis rosea; and that in its later, infiltrated, serpiginous, and at times moister stages psoriasis is quite easily confounded with syphilis and with

lupus vulgaris. Such possible mistakes naturally entail serious therapeutic and prognostic blunders.

Concerning the treatment of this at times almost hopelessly intractable disease let me make two suggestions: There is a multiple, highly inflammatory type of eruption where all of the usual, exciting, detergent drugs serve only to add fuel to the flames. Here we can apply a mild ointment and frequently derive rapid and gratifying benefit:

℞.	Bismuth. subnitrat.	2
	Acid. boracic.	4
	Ol. olivarum.	8
	Lanolin	24

More frequently we are confronted with old, desperately chronic, deeply infiltrated, thickly desquamative plaques which cover large areas of the trunk and limbs. Preceding medical attendants have tried numberless medicaments. In such cases we at the Massachusetts General Hospital, at the instigation of Dr. Loretta Joy Cummins, have instituted the carbohydrate-poor diet, applying to the surface only the most perfunctory medication. We still have our failures in these trying cases, but we feel assured that their numbers have been decidedly diminished since we have perfected this method.

This radical procedure is not advisable in growing children, on account of the possibilities of an ensuing acidosis and because we consider the natural growth of the child more important than the incidence of psoriasis. We do not advocate this selective starvation for the thin type of adult. Therefore, bearing in mind the possibilities of acidosis and of too great loss of weight, we begin the treatment. We give each patient printed sheets of the Allen diabetic diet and recipes for the making of bran and agar cakes and bran and Lyster flour muffins and doughnuts. We insist on accurate weight charts from each patient and we stop the diet if it is found too depleting. In the first week there is always a loss of 3 to 4 pounds, and in the succeeding three or four weeks a further weekly loss of perhaps 2 pounds more. Then equilibrium is apt to be established and from then on there is usually no further loss.

This little-known method may seem decidedly bewildering, for previous dietetic investigators have strongly urged the elimination of protein from the daily bill of fare. My only answer is, try for yourself this more recently advocated method. My only explanation of these contrary teachings is that, after all, perhaps the value of either lies in the semistarvation of the patient.

PITYRIASIS ROSEA

Diagnostically, let me emphasize two very frequent errors. The first is the mistaking of this disease in its early, undifferentiated, macular stage, for syphilis—naturally a very serious blunder, but, unfortunately, in my experience, not at all a rare one. The second, even more common, is the confounding of this disease with multiple tinea circinata. The fault generally lies in forgetting that there is such a malady as pityriasis rosea. Bearing constantly in mind the existence of this harmless affection will easily obviate these unfortunate errors, and the use of the microscope and the Wassermann test will render the proper diagnosis a comparatively easy matter.

Therapeutically let me warn against the use of strong applications in this affection; they are not only unnecessary but also distinctly harmful. It is not an exaggeration for me to state that in my experience the great majority of patients with pityriasis rosea have been overtreated by their first attendants, and come to me with lesions so inflamed that the proper diagnosis is often a matter of great difficulty. Generally, the first physician has considered the outbreak to be ringworm, and has prescribed tincture of iodin, mercury in some form, or sulphur, and the result is a weeping or semimoist dermatitis venenata. Whatever our beliefs may be concerning the etiology of the disease, the fact remains that pityriasis rosea is best treated by means of soothing applications; and in my own hands "white wash" (*vide* Erythema Multiforme) in summer and "pink wash"

℞.	Acid. carbol.	1.30
	Calamin	4
	Zinc. oxid.	8
	Glycerin	8
	Aq. destillat. q. s. ad.	250

in winter have proved entirely satisfactory from a curative and from an esthetic point of view. Winter, in the climate of Boston, means the period from September 15th, to June 1st; summer, from June 1st to September 15th. These washes should be sopped on whenever and wherever subjective symptoms arise; the diet should be "light"; the clothing should be non-irritating and loose; work, especially in warm weather, should be kept down to a minimum; and a starch bath (*vide* Urticaria) should be taken, if acceptable, once a day.

ECZEMA

The diagnosis of this common disease is usually comparatively simple, save in the palms and soles, and in these areas the general practitioner must be on his guard lest he confuse this disease with syphilis, or with psoriasis, or with infection by the Epidermophyton inguinale, a relatively unrecognized variety of the ringworm plant. Syphilis and psoriasis of the palms are adequately described in the text-books and should require no reiteration here, but the clinical appearances of Epidermophyton inguinale infection have not been as yet sufficiently elucidated except in the very latest editions of dermatologic treatises, and may very properly be detailed at this juncture.

Most of us are acquainted with this plant in connection with the sharply demarcated, pink-red, delicately scaling homogeneous plaques found on the inner upper thighs, next the pubic arch and, usually on the left side in men, because of the contiguity of the scrotum. We have called this "red flap," eczema marginatum, jockey-strap itch, etc., but now, thanks to Sabouraud, of Paris, we know this to be due to a certain type of ringworm. More recently we have learned that similar conditions may arise over the pubes, around the umbilicus, in the axillæ, along the perineum, between the nates, and in the bend of the knees and elbows. Let us, therefore, not forget these pitfalls, for we may treat these infections as eczema for an indefinite period and get no results; whereas a proper diagnosis plus the application of Whitfield's (of London) ointment will ensure a cure in these areas in a comparatively few days:

℞. Acid. salicyl..... 2
Acid. benzoic..... 4
Adipis benzoat..... 30

Within the last two years we have learned that this same plant, the Epidermophyton inguinale, may produce lesions on the palms and soles, between the fingers and toes, and, to a lesser extent, on the contiguous dorsal surfaces of the hands and feet. To simplify the description of these relatively unrecognized infections let us divide them into the dry and the moist types.

Dry Type.—Between the toes, for instance, we may be confronted with stratified scaling, which, at the bottom of the space, where heat and moisture are the greatest, may take on the consistency and color of lard. This condition is apt to be most prevalent (for the reasons just given) between the fourth and fifth toes. Questioning will reveal the fact that this desquamative condition has probably persisted for months and even for years, and that in most cases, for obvious reasons, it is most conspicuous in the warmer months of the year, often in winter dying down into almost nothing. From these one or more foci we may note a delicate redness and roughness and desquamation extending outward and backward, always with a fairly well-defined circular border, on to the plantar surface of the foot or more rarely on to the dorsal face. The exact prototype of this clinical picture may and does occur between the fingers (but always, naturally, to a lesser extent) and on the palms and backs of the hands, where the condition may be more extensive and more intense than on the feet. Itching is usually present and may be violent.

Moist Type.—Here we are dealing more with the lateral aspects of the fingers, the middle of the palms and of the soles, and especially with the arch of the foot. The horny layer in these parts (except that of the digital sites) is decidedly thick, and we note first a deep, seemingly intracutaneous vesicle which sooner or later becomes semipurulent, reaches the surface, breaks, and often forms a minute opening surrounded by a curious, collar-like scale. These primary vesicles are apt to

form in groups, and the resulting phase produces a strikingly cribriform area with diminutive serpiginous outlines. In earlier years we have called this condition dysidrosis and dermatitis infectiosa eczematoides, and possibly dermatitis repens. It is not my intention to claim that these diseases do not exist, but it is my present purpose to emphasize the fact that the Epidermophyton inguinale can be demonstrated in numerous examples of these hitherto frequently improperly diagnosticated skin eruptions.

When once the correct diagnosis has been made it is a pleasure to both patient and physician to observe the rapid subjective and objective relief from what has, up to this moment, proved an intractable and perhaps even cripplying condition. This rapid change is produced by the inunction of Whitfield's ointment (*vide supra*), by the daily washing of the affected parts, and by the sterilization of gloves and stockings.

From a therapeutic point of view of eczema I want to speak of two types—the infantile and the winter varieties—of the disease.

Eczema generally attacks babies in one of two ways. As a rule, the moist form predominates and consists of redness, edema, excoriations and crusts on the face and forehead, and, to a lesser extent, on the scalp, buttocks, hands, and flexures. Relatively occasionally we meet the dry, slightly infiltrated, somewhat scaly type of the disease. In the latter case the eruption is liable to be on any portion of the body.

The introduction of crude coal-tar (*vide* Lichen Simplex) has, in my own experience at least, revolutionized the treatment of infantile eczema of the wet type. Before its introduction one could count on two winters and an intervening summer of treatment. One was obliged to insist on the use of masks and the constant fastening of the victim's hands to the bed. The mother or attendant nurse required an extra pair of hands with each change of the child's dressings to prevent severe damage to the uncovered skin—more damage than could be repaired during the succeeding interval of treatment; and despite all these cares and anxieties the child remained in the greatest

discomfort and the disease failed to disappear. Now the picture has changed. The constant exhibition of crude coal-tar, employed in the proper manner, within a few hours usually controls the itching, and within a comparatively few days cures the disease in the majority of cases. There must, of necessity, be exceptions to this rule. Then we insist on a microscopic examination of the patient's feces, which in these wet cases usually reveals the child's inability to digest its fats. We then naturally reduce the ingestion of fat or of starch, if that be the case, and proceed with the crude coal-tar as before. Even then a few recalcitrant examples persist, and in such an event we make cutaneous food tests, when finally we may discover the noxious etiologic agent. Unfortunately, the dry cases of infantile eczema hold out no such rosy outlook. Here crude coal-tar is utterly unsuitable and no genius has found its counterpart.

The treatment of winter eczema is not difficult when one bears in mind the fact that the redness and the delicate scaling and the itching are due to the dryness of the atmosphere, which begins in the latitude of Boston about September 15th, and continues well into the spring. Apart from the dryness of the atmosphere we live frequently in houses heated by hot-air furnaces, we take hot baths, we use drying soaps, and many of us, both men and women, no longer wear long-sleeved and long-legged underclothes.

To counteract these ill-effects let us lay down certain definite rules for every-day life, and let us warn our patients to begin to observe these instructions early in the autumn when dog-days have left us and the cool, invigorating northwest winds begin to blow. Have pans of water on every radiator and keep the furnace water-boxes full. Cultivate as many growing plants in the living rooms as possible. A starch bath (*vide* Urticaria) should be taken every night, using a superfatted cold-cream soap. Wear tight-fitting sleeves to the shirts and long legs to the drawers. Wear no silk or wool next to the skin, but, if wool is necessary, let it be worn outside of cotton. Before dressing in the morning, after undressing at night, and whenever the skin itches or tingles or burns apply warmed "pink wash" (*vide*

Pityriasis Rosea). If there is an appreciable amount of infiltration and scaling, a distinctly greasy, mild ointment is very grateful:

℞. Bismuth. subnitrat..... 2
Ol. olivarum..... 8
Lanolin..... 24

Severely intractable cases of winter eczema, especially in old people, are not always cured by these methods, but analgesic doses of *x*-rays from an expert's hands practically never fail to relieve and to cure this troublesome condition.

HERPES SIMPLEX

Many a golden opportunity has been lost in confusing "cold sores" and the initial stages of hard and soft chancres. There is only one maxim to follow: When in doubt, take a smear and examine it under the dark-field lens or stain this smear and study it under the oil-immersion lens. Ignorance or carelessness in differentiating these frequently confronting clinical similarities are grave offenses against a patient's rights.

One is often consulted by individuals who suffer from recurrent attacks of herpes on the lips or the cheek or the genitals. In such cases the long-continued ingestion of hexamethylenamin has not infrequently eventually stopped this annoying condition—5-grain tablets dissolved in a whole glass of hot water and taken after each meal for perhaps two months, and then less and less frequently for many weeks, have proved efficacious in my hands in more than one instance.

HERPES ZOSTER

A word of caution here as to the diagnosis of this disease. Quite often a neuralgic stage may precede by some days the actual cutaneous eruption. Patients have been treated by physicians merely for their nerve disturbances, and no cutaneous examination has been made. One rather remarkable instance recurs to my mind. A lady consulted an aurist for pain about the right ear; in a few days she consulted an oculist for a similar

condition of the right eye; then she asked the assistance of a medical man because of a right-sided headache; then she went to a surgeon because of pain in the right side of her neck. The surgeon asked my opinion, and inspection revealed a few groups of rather abortive vesicles scattered through the thick hair on the right side of the head. This, of course, is an extreme case; but not at all rarely does one find an obscure, antecedent neuralgia, the nature of which is eventually divulged by the subsequent characteristic eruption of grouped vesicles.

Don't let us forget that freezing the seat of the affected nerves twice daily with ethyl chlorid will cause rapid relief from the painful accompanying neuralgia, and will, in all probability, ward off the frequent and long-continued neuralgic after-pains which are especially severe in elderly people.

POMPHOLIX (DYSIDROSIS)

Whenever confronted with these closely allied conditions be sure to rule out the possibility of an Epidermophyton inguinale infection. Plant some of the fluid on Sabouraud's medium,

℞.	Peptone	0.50
	Agar	1.30
	Maltose	3.80
	Water	100

or examine the fluid under a high-power lens, excluding much of the light. Let me warn you, however, that the presence of this hyphomycete is never easy to demonstrate.

IMPETIGO CONTAGIOSA

The general practitioner recognizes this disease in most instances, but there are two varieties of the infection which are very apt to lead one astray. Occasionally in the newborn we meet with an abundant bullous eruption about the nates, and less often around the genitalia. In my experience this condition is practically always called syphilis, sometimes pemphigus, almost never impetigo contagiosa. Nevertheless it usually is impetigo contagiosa and strict asepsis plus an ointment of

℞. Acid. salicyl..... 1
Sulph. præcip..... 1.5
Adipis benzoat..... 30

will usually cure these really sick babies. The second confusing type of the disease, and one which seems rarely to be diagnosticated properly, is the circinate form where one notes large round circles of crusting moisture, sometimes in concentric forms. This is a rare variety, but it yields equally well to antisepsis and the application of a suitable ointment.

As to the treatment of the disease in general, my criticism would be that the presence of pediculi capitis has not been duly considered; that sufficient stress has not been laid on antisepsis (the key to the whole success of its management); that the barber has been allowed to continue his various ministrations without proper instructions; and that too strong applications have been employed. Gentle shampoos with powdered soap; drying with fresh, sterilized gauze; freshly boiled pillow-cases and sheets and handkerchiefs every day; repeated washings of the hands; the use of fresh gauze instead of shaving brushes, and of powdered soap and of boiled razor-blades in shaving; and the constant use of one of the following ointments will invariably prove successful within a week or ten days:

℞. Acid. salicyl..... 2
Sulph. præcip..... 2
Adipis benzoat..... 30

or

℞. Hydrarg. chlorid. ammoniat..... 0.13
Acid. salicyl..... 2
Adipis benzoat..... 30

DERMATITIS VENENATA

This important disease seems to provoke the greatest number of pitfalls for the unwary practitioner both in diagnosis and in treatment. To be a master of this common, trying, and not infrequently crippling condition one should have the detective powers of a Sherlock Holmes and the most intimate knowledge of the world's irritants, whether of the animal, vegetable, or

mineral kingdoms, whether used or encountered in work or at play or in mere existence.

It would be impossible here even to enumerate the world's noxious substances or the various trades, occupations, pastimes, and practices which expose one to them; suffice it to say that they are endless, but that, fortunately, most of us are immune to their influence. Nevertheless, the victims are many, and it should at least be our duty to recognize them at the very first opportunity and to treat them properly. I regret to say that we who practice medicine are not all experts in this special branch of medical knowledge—far from it in fact. In the first place, don't call this disease erysipelas, and second, try to master the special features which separate this affection from eczema. The differences are very subtle at times and perhaps impossible wholly to put on paper. However, when you observe a rather indefinite, possibly blotchy redness, and particularly even a tendency toward vesiculation, especially when these lesions are accompanied by itching and occur on exposed places, always suspect some external irritant, and begin at once to ply your patient in the most inquisitive manner concerning his occupation, his habits, his customs, and his pleasures. Bear in mind especially the possibility of poisoning by furs, hair dyes, poison ivy, and the poisonous primrose.

In regard to furs, remember that it is toward spring when the neck perspires that the dye is most apt to be dissolved. Therefore don't be led astray by the remark that "it can't be this (coat or this boa), because I have worn it all winter." Be on the lookout for lusterless hair and don't be afraid to ask questions, although one must use tact in dealing with this type of patient, for people who dye their hair are apt to be "touchy." Remember that the smoke from bonfires containing the wood of poison ivy is particularly potent, so don't exclude this type of poisoning even in winter. Finally, don't forget the existence of the poisonous primrose, usually the Primula obconica, for in winter this plant plays a large rôle in the production of dermatitis venenata. I shall always remember one consultation. I was called to a neighboring town to treat a case of "recurrent

erysipelas." On entering the patient's room I found her sitting beside a table covered with these insidious plants. The "recurrent erysipelas" was easily cured. It used to be an easy matter to identify this plant, for in its original guise it consisted of a relatively large olive green leaf the shape of the ace of spades, and covered on its under side with many whitish hairy spines; of a long stem covered with spines similar to those on the leaf; and of a cluster of ten or twelve small, delicate, pale lavender flowers. Nowadays this description holds true only of the hairy spines, for through the manipulations of the florists the leaf is of a lighter color, the stems are not so long, and the flowers are considerably larger, and may be pink or red.

Let us consider dermatitis venenata from the point of view of location. If the ears and forehead and scalp are affected, think of hair dyes or the possible application of kerosene. If the neck and upper chest are involved, ask about furs, and remember that the suspicious ones are usually those which lack luster and are dead brownish or dead blackish in color. If the trouble lies in the axillæ and their neighborhood, inquire into the question of depilatories and preparations against perspiration. If the pubes and the penis are inflamed, look for pediculi pubis, and query the patient about the use of kerosene and especially "blue butter." If the penis and scrotum are red and edematous, suspect poison ivy. When the disease affects the face and hands no immediate inference can be drawn, because these necessarily exposed surfaces may come in contact with almost any conceivable irritant, according to the special circumstance.

The treatment of dermatitis venenata should be a very simple question, and if the cause can be determined the cure should be possible within a week or two, granted, naturally, that the proper measures are advised by the physician and strictly followed by the patient. Past experience, however, has demonstrated very clearly that treatment in very many cases has not been so successful as it should be.

The importance of this subject and the possibility of easy and prompt success warrant a thorough exposition of at least

one efficacious method of treatment. It is assumed, of course, that the cause of the dermatitis has been thoroughly investigated and determined if possible. The patient is first advised to take a bath, which should include the scalp, face and hands, and whole body, using plenty of soap and rubbing down afterward with alcohol. This precaution will probably do no harm, and may prove very successful in aborting subsequent development of the disease in other parts of the body. The patient is then advised to stop work and to stay at home in loose garments; to live on the lightest and most digestible diet; to take a diuretic,

℞. Potass. acetat..... 32
Aquæ..... 250
Sig.—A tablespoonful in a whole glass of water t. i. d., p. c.

to keep the bowels freely open; to sop on "white wash" (*vide* Erythema Multiforme) every hour (avoid all bandages) until the skin looks and feels like a whitewashed fence; and to drop into the eyes, if the lids are closed, a 10 per cent. solution of argyrol.

This stage of the treatment should not require more than a week, possibly not more than two or three days. When the skin is stiff and hard and even cracked, and is oozing serum, then begin the second stage of the treatment—the process of repair. Apply "pink wash" (*vide* Winter Eczema) twice a day, and as soon as it has dried smear on Lassar's paste (*vide* Erythema Multiforme). This should be covered with one layer of thin old cotton or linen cloth fastened by safety-pins. This second phase of the treatment usually requires only a week or somewhat more. Contrast this rather seemingly rosy prognosis with the possibilities of maltreatment. From a rather long consultant experience, in which more than a few instances of unsuccessful treatment of dermatitis venenata have been encountered, two especially striking cases recur to my mind. A man said that he had been poisoned by ivy two weeks previously, and presented what looked like an extraordinarily large arm. The attending physician had made some greasy applications and then had applied

yards and yards of bandage and enclosed the whole with oiled silk. On taking down this elaborate dressing the stench was almost intolerable, and the whole arm was in a state of intense edema, lymphangitis, and superficial gangrene. The second example refers to a gentleman whose skin was poisoned and subsequently, unfortunately, maltreated, so that eczema supervened, and the hapless victim was unable to attend his daughter's wedding more than a year later.

Before your patient leaves you warn him that for a few weeks the skin will be very susceptible to infection, and to cold, heat, and friction, for there can be but little or no horny layer on the affected parts. Also bear in mind that for a short period or for years the individual's skin has become sensitized to the original irritant and to its congeners. Let me illustrate this point as evinced in a recent case of bromin-poisoning. A lady presented a small but foul ulcer near the ankle. Ordinary antiseptic washes and ointments, even with rest in bed and elevation of the foot of the bed, made no impression on the sluggishness of the ulcer. An oily preparation of Dakin's solution was then applied, and almost immediately the size of the ulcer began to increase and the pain to intensify. In a casual remark the patient stated that she was taking sodium bromid from another physician! Here then was the effect of one irritating drug, chlorin, on its original congener, bromin. Both drugs were stopped, and the sloughing character and the rapid extension of the process ceased at once, and the wound progressed with gratifying celerity to a perfect healing.

DERMATITIS MEDICAMENTOSA

This subject is too large to describe in detail here, but nevertheless it is included in this survey of common errors because the general practitioner seems at times to fail to bear its possibilities in mind, and to ignore the fact that mother's milk may convey to the nursling the exciting drugs that she may be taking at the time. After listening to the above anecdote one might very properly accuse me of just this fault. The only excuse would be to say that the patient presented none of the

characteristics of chronic bromin-poisoning, *i. e.*, no mental torpor, no dull facies, no acneiform lesions of the face and shoulders, and no tendency in the ulcer toward papillomatosis. There seems to be only one sure road toward the mastery of this complicated and important chapter in dermatology. First, one must learn by rote the possible changes in the skin induced by the known mischief-making drugs; and second, one must recognize the fact that the objective lesions presented may not conform to those of other well-recognized diseases. Then one must begin to ask questions; but here again, as in dermatitis venenata, one must not be contented with "No" for an answer. Persistency must be our watchword. Some patients do not think; some don't want to, and others will deliberately lie.

DERMATITIS FACTITIA

Text-books are perhaps more apt to speak of this condition as malingering or again as hysteric gangrene, but the above title seems, on the whole, the best one. I shall try to make myself very emphatic and very explicit on this subject, for the errors I have seen committed in this disease, even by some of the best surgeons, can only be described as butchery, and, curiously enough, when the error has been pointed out to them they frequently refuse to believe one word they are told. The Skin Department of the Massachusetts General Hospital has a striking series of photographs illustrating what these patients have begun, and, in some cases, what too credulous surgeons have finished.

The only way to recognize this condition is: first, to appreciate that it exists; second, to be struck by the fact that its main features cannot be explained by ordinary disease; third, not to be led astray by sentiment or by specious arguments. Never mind if a schoolteacher tells you with tears in her eyes that she is poor and must return to work as soon as possible; never mind if a good-looking young girl has great ulcers on her breasts; never mind if a young girl has allowed one joint after another of a finger to be cut off; never mind if a woman has allowed tissue to be excised from her chest and abdomen to such

an extent that the resulting scars suggest nothing short of an Armenian massacre. Despite all these seemingly convincing arguments against what might be called common sense, don't waver when once you have made up your mind that dermatitis artefacta is present. Don't let even your elders persuade you from a proper diagnosis. Above all, pay no attention whatever to what the patient may tell you. These victims are practically always hysteric and may commit these self-mutilations in a subconscious state. These sentences may seem dictatorial, unsympathetic, arrogant; call them what you will. I can merely repeat that the Skin Department of the Massachusetts General Hospital has had a large experience in this condition and knows whereof it speaks.

DERMATITIS ACTINICA

As time goes on we recognize more and more fully the capability of good and of evil exercised by the Roentgen rays. It has been my privilege often to prescribe successfully the *x*-rays in the treatment of several obstinate dermatoses, but it has also been my lot to see the cruel results of too long and too frequent exposures to these same rays. Nowadays carelessness in screening and a too rapid repetition of the exposures usually produce the evil consequences. Quite a series of patients have consulted me for alopecia following the application of the rays to determine the presence or the absence of dental cavities. Far too great a number of luckless individuals have come to me for the treatment of *x*-ray burns of all degrees. Let me advise you, therefore, to test the susceptibility of each individual before undertaking any prolonged or repeated exposures; let me warn you to screen properly the actual focus and all surrounding tissue before turning on the current; let me, finally, ask you before making the exposure to remove with some solvent all grease from the skin, and not to use on the exposed surface any grease whatsoever for a period of ten days at least after the exposure. Grease beforehand materially alters the penetrability and the intensity of the rays; grease afterward, in case of hypersusceptibility or overexposure, materially "makes a bad job worse."

If a patient consults you for an *x*-ray burn, remove all grease at once, forbid its future use, and prescribe "white wash" (*vide* Erythema Multiforme), to be sopped on every hour, or whenever the pain demands it, and never bandage. Within a few days it will be evident whether or no you have a superficial and an amenable case to deal with. If the pain and redness and edema don't subside, if an ulcer is present or soon develops, don't waste another moment on external medication. There is only one proper and successful method of relief from the exhausting pain and the intractability of the lesion, and that lies in surgery—a wide and deep excision of the whole affected area and a subsequent skin-graft. Long experience has firmly convinced me of the truth of this statement.

LUPUS VULGARIS

Lupus vulgaris is not a common disease in New England, and yet it leads to no end of mischief in the hands of the medical unwary. In the Middle West and in California this disease is frequently confounded with blastomycosis and with oïdiomycosis. Clinically, we must bear in mind that the skin in blastomycosis is usually riddled with pus canals from which the yellow fluid can be easily expressed, and that its periphery is almost always of a marked purple hue. The similarity between lupus vulgaris, on the one hand, and blastomycosis and oïdiomycosis, on the other, however, may be so great that a resort to the microscopic examination of the pus and of the stained sections and even to cultural methods may be obligatory before a positive diagnosis can be made and accepted.

To the New Englander this differentiation is not the usual pitfall. The eastern medical man must be forever on his guard that he does not confuse lupus vulgaris with syphilis, and vice versa. It is no exaggeration for me to state that this unhappy mistake is not at all a rare occurrence, and this is especially true in the case of the nose; in truth, it seems at times as though certain practitioners could not be aware that there is such a condition as late syphilis of the nose. Syphilis of the nose is apt to be found after the age of thirty; as a rule lupus occurs in

childhood and in early adult life; syphilis presents a turgid red-blue color, lupus is apt to be a brownish red; syphilis is prone to appear as a rather homogeneous swelling; lupus usually presents one or more so-called lupus nodules; syphilis is less crusted than lupus; syphilis is more frequently accompanied by the odor of necrosis than lupus; there is more variation in the intensity of the syphilitic process; lupus is apt to run an unchanged course. I enter especially into these details because I have witnessed sad mutilations of this most conspicuous feature when a better knowledge of skin diseases, or a little more study of the individual case, would have enabled the practitioner to have administered salvarsan or iodid of potash and saved the integrity of the face. There are, of course, confusing examples of the two diseases in other parts of the body, but these mistakes are less disfiguring, although the consequences may be equally disastrous in the long run. Be on your guard, therefore, and remember the fact that these two diseases may often bear so close a similitude that serologic and therapeutic tests must be invoked before a final and definite conclusion can be drawn.

ERYTHEMA INDURATUM

This is a disease which is not as well understood as it should be, and when the physician is confronted with the one or more deep, subcutaneous nodules he is apt to consider the disease syphilis and to administer iodid of potash, a drug which usually causes a marked exacerbation of the hitherto sluggish condition. When called upon to diagnosticate this subvariety of tuberculosis remember that the gummata of syphilis when on the legs are apt to be single; to occur over some part of the front of the tibia; to arise with some rapidity; to appear in middle life or later, and to be asymmetric. Erythema induratum, per contra, usually presents several or more slowly evolving tumors, at first deep in the panniculus adiposus, and gradually extending to the surface; the nodules are on the back and sides of the legs and both legs are apt to be symmetrically involved; the patient is almost always a young woman and one in whom tuberculosis may be present. It will not be amiss to detail in this connection

a form of treatment which is but little known and has, as yet, found no place in the text-books. We owe this successful method of treatment of this hitherto intractable malady to Dr. F. S. Burns, of the Skin Department of the Massachusetts General Hospital. The proçedure is simple and consists merely in the thorough curetage of each lesion down to the fascia, allowing the wound to granulate up from the bottom, with subsequent skin-grafting, if necessary. The operator will be struck by the depth and the extent of each nodule and by the unexpectedly short period of convalescence, granted that an abundance of fresh air and easily digested and nourishing food are obtainable.

SYPHILIS

Medical literature and personal experience are so full of errors in the diagnosis of syphilis that it seems hardly necessary to enter into details here. Suffice it to say that we must always be on our guard lest we confuse primary lesions with herpes progenitalis, "soft sores," carcinoma, felons, boils, and gummata. One is particularly liable to error in extragenital chancres, especially when near the finger-nails or on the tongue or the tonsil. In the early secondary stage of the infection we must not confuse the roseola with drug eruptions or the exanthemata or pityriasis rosea; nor the papular type with lichen planus, follicular eczema or dermatitis medicamentosa; we must not mistake hemorrhoids for condylomata; we must not call the patchy loss of hair alopecia areata; we must not fail to note the significance of mucous plaques. In the later seconday stages we must be very careful to differentiate multiple ringworm, psoriasis, and lupus vulgaris from the serpiginous types of the disease. In the so-called tertiary stage we must watch out that we do not mistake gummata for carcinoma, erythema induratum, or perhaps mycosis fungoides. In other words, throughout our whole career we must remember that syphilis is the great "imitator," and that if we cannot all have a special knowledge of skin diseases, we can at least own and become proficient in the use of the dark-field lens and in the taking of blood for the Wassermann test. These two procedures alone, if practised universally,

would eliminate the great majority of physicians' many errors in this truly large and important field of medicine.

TINEA TRICOPHYTINA

From a differential diagnostic point of view I have already spoken of ringworm under several previous headings; but there is one type of the disease which is but little known and which I have not mentioned, and that is the multiple purulent infection of the hairy regions. This infection is due to the variety of the ringworm plant known as the Megalosporon ectothrix—a variety derived from horses and from cattle. When this specific infection occurs in the scalp it is called kerion Celsi, and consists of a definitely round, sharply raised plateau, the surface of which is red and raw and riddled with holes (the hair-follicles) from which pus runs, or else is confined within a pustule. This phenomenon is known and recognized by a certain proportion of general practitioners, but its analogue in the beard seems to be but little known to any save the expert. In the beard the infection is not confined within any definite limits. The process is of very rapid evolution, and in the space of a few days the patient notes one follicle after another involved in intense swelling and inflammation and literally streaming with pus. Don't mistake this for an ordinary sycosis, it is not. Fortunately, a cure is not at all difficult, principally because the hair, the food of the plant, is detached from its papilla and is floated off in the stream of pus. Hot water fomentations three times a day and the inunction of the following ointment will bring about a cure within a few weeks:

℞. Beta-naphthol 2
Acid. carbol. 4
Sulph. flor. 4
Adipis benzoat. 30

Don't bandage.

From a therapeutic point it will not be amiss to emphasize here the great value of the *x*-rays as an epilating agent in the treatment of the ordinary ringworm infection of the scalp.

When we remember that cure of this trouble by our older methods necessitated exclusion from school for a period of six months and that it entailed the constant use of ointments we can well appreciate the boon conferred on school children and their parents by Sabouraud's introduction of the *x*-ray method. No tyro should undertake this work. The dangers of an *x*-ray burn or permanent baldness demand absolutely the expert's care in these cases. Few of us are experts in radiology, but that should not deter a single one of us from sending every case of ringworm to the expert, no matter how far away he may live, granted that such a procedure is feasible. One exposure suffices, provided that the infected area is not too large. Within three to six weeks new hair, free from the previous organism, begins to grow, and the child can then go to school. Remember the injunction about the use of any grease whatsoever within a period of ten days after an *x*-ray exposure. After this danger period apply twice daily to the affected surface the ointment recommended for the Megalosporon ectothrix (*q. v.*). These inunctions will keep down any possible superficial reinfection and will stimulate the growth of the new hairs.

KERATOSIS SENILIS

We are all familiar with the rough pigmentary patches on the face and on the backs of the hands of individuals. We must not confuse this disease with vitiligo, with chloasma, or with xanthoma—keratosis senilis is accompanied by hyperkeratosis. Nor must we exclude this diagnosis because the affected individual is still young or because he has not led an out-of-door life. The disease may exist in early adult life and even in "shut-ins." Let me ask you here not to waste time on ointments. They do not penetrate deeply enough to effect a cure. Radium or the more rapid curet plus subsequent cauterization are essential, and when properly used produce a permanent cure. As to our choice of procedure, remember that radium is less painful and will produce the better cosmetic results; but the curet is quicker and cheaper. Explain the alternatives and abide by the patient's decision. Before dismissing your patient explain fully

the etiologic effect of strong light on the development of future lesions, and tell him that before any prolonged necessary exposure to light in the future it would be well to cover the face and hands with "pink wash" (*vide* Pityriasis Rosea) in winter and "white wash" (*vide* Erythema Multiforme) in summer.

WARTS

Verruca Vulgaris.—It has been my lot to see a good many young people, especially girls, affected with warts—ordinary "seed warts." These patients ask what is the best form of treatment by which they may be rid of their disfiguring "blemishes." They very often say that they don't want any more warts burned out because the resulting scar is so ugly. This is the point I wish to emphasize. Don't burn warts with acids or the thermocautery, for the scars are ugly. In my opinion it is better to do nothing than to leave permanent disfigurement, which, after all, is not usually necessary, because warts on the hands will almost inevitably eventually disappear of themselves. Assuming that verruca vulgaris is produced by an animal parasite, an antibody is produced sooner or later which renders its life tenure impossible. Such a biologic phenomenon will properly explain the thousand and one "cures" for warts. However, the average patient does not care to be told by a physician to leave things alone. He consults a doctor because he wants positive help and action. What advice can one give, then? I believe we have a solution of this old question in the high-frequency machine, and, in my opinion, fulguration (as the method is called) in the expert's hands will practically always cure the disease within a reasonable time. Curiously enough, as you probably know, the operator does not have to electrocute every lesion, for after a certain number have been treated the remainder often suddenly disappear *en masse.* Here again antibodies are presumably at work.

This method of fulguration is especially useful in warts of the sole of the foot—hitherto a really serious therapeutic problem. Don't excise warts in this part of the body. Sterilization of the field of operation is peculiarly difficult; the procedure

is always temporarily crippling; and very often the wart returns.

Verruca Plana Juvenilis.—This is a variety of wart which is not well understood. It exists on the face and on the backs of the hands—exposed places—in the form of one or many, yellow-buff, or occasionally pink, flat-topped, squarish or oblong, smooth-surfaced, scaleless papules, which often occur in linear groups. These marked objective divergencies from other varieties of warts cause one to wonder why these tumors should be grouped under the general heading of verruca until we find that etymologically the word means an eminence. In the refinements of intricate diagnosis we must not confuse verruca plana juvenilis with some of the multiple benign cystic epitheliomata—syringoma or syringocystadenoma, for instance; otherwise bearing in my mind the position, the number, the color, the character of the top, the shape and the distribution of the lesions, the diagnosis is simple.

This not very common tumor has been included in this lecture because when present in abundance it may ruin temporarily a sensitive person's life, and because there is, in my opinion, a definite cure for it—a cure which has not yet been accepted by the text-books, but one in which we at the Massachusetts General Hospital have faith. This cure consists in the internal administration of the following pill:

℞. Hydrarg. iodid. virid..... 1
Ext. gentian..... q. s.—M.
Ft. pil. No. lx.
Sig.—For an adult, one pill t. i. d., p. c.

The disappearance of the warts, following the ingestion of these pills, may take place within two weeks, but more often the tumors are more obstinate, and in certain instances several months may be required to effect a cure. Don't lose heart under these circumstances. Be persistent, and in the end you and your faithful patient will be rewarded. During these weeks or months of mercurial ingestion never forget the strictest oral antisepsis.

Verruca Senilis.—This is, of course, a well-known variety of wart, but one which may require surgical interference. Tell your patients to watch these tumors, and if they grow unduly large or take on unaccustomed hyperkeratosis or bleed, advise radium, careful excision, or curetage and cauterization.

PRURITUS

Pruritus senilis is one of the banes of old age, but the well-informed physician can assure his patient a perfect cure. Innumerable applications have been recommended and usually in vain, for none are successful beyond a certain point. The *x*-rays, however, when administered by an expert in so-called analgesic doses, are invariably successful, and constitute, so far as I know, the only method of cure. Certain details of living will aid in this cure, and have been described in this lecture in connection with the paragraphs on winter eczema (*q. v.*). It is assumed, naturally, that a previous careful examination of the cardiorenal functions of the patient has been made.

Pruritus vulvæ and pruritus ani are likewise two great dermatologic problems. The solution of these vexing questions was never wholly solved in my opinion until the discovery of the therapeutic uses of crude coal-tar. Volumes have been written on the subject of the treatment of pruritus ani. Divers drugs, internal and external, have been tried and found wanting. Various operations have been described and performed, many times unavailingly. Elaborate physical examinations, never to be deprecated, of course, have been instituted, but usually without results. The cure, however, as hinted at above, is really very simple, and what I am about to describe is based on personal experience, even of desperate cases which have persisted over many years, and have defied the care of the most skilful physicians, surgeons, and proctologists.

In the case of pruritus ani it is assumed that personal hygiene to the uttermost will be explained and enforced. To be explicit, personal hygiene includes the maximum amount of fresh air and sleep; the proper proportion of work and play; the devotion of a certain amount of time to daily out-of-door exercise, according

to the age and ability of the individual; a daily bath of a temperature best suited to personal temperament; the ingestion of simply cooked, easily digested, well-balanced food, slowly eaten and followed by a half-hour of mental and physical relaxation; the avoidance of all stimulants, solid as well as liquid; and finally, the daily easy movement of the bowels. Such a regimen is a *sine qua non* to the best and quickest results.

The hygiene of the anus is of paramount importance. Experience has shown that pruritus ani is almost invariably accompanied by redundancy of the anal folds, and the depths and recesses of these folds serve as hotbeds of bacterial life, and consequently must play a decided rôle in the production and continuation of eczema and itching about the anus. Therefore we must counteract these mischievous anatomic defects as much as possible. Don't hesitate in this tormenting condition to go with your patient into the minutest detail of treatment. "The art of taking pains" should ever be a maxim with the dermatologist. After defecation the patient should remove the external fecal remainder with wet, thin toilet paper. He should then get into a squatting position on the floor with a good light and a mirror before him and should dry the anal region most carefully and thoroughly with absorbent cotton. Remember that heat, darkness, and moisture favor bacterial growth. Still remaining in this squatting position, with the mirror on the floor before him, the patient should apply to the interstices of the anal folds, by means of a fine cotton swab, a minimum amount of a 5 per cent. crude coal-tar ointment. The patient should not adopt a careless, forward and backward movement of the applicator, but should begin always at the lumen and paint outward to the periphery of the local pigmented area. The painting of the spokes of a wheel would be a somewhat similar proceeding, only in this instance we are painting in between the spokes. This maneuver should be performed morning and night, and before each fresh application the old ointment should be carefully removed with the oil of sweet almonds. Remember, as in all cases where crude coal-tar is to be used, to clip the neighboring hairs and keep them short.

Such is a successful method of treating pruritus ani, and, if pursued *in toto* by you and your patients, I believe will not often fail you.

This brings me to the end of what I want to say to you today. The omissions and commissions which I have endeavored to describe do not comprise single instances—they have been numerous. I have thought of them often and wondered how they could be avoided. For this reason I have come before you today, hoping by honest, constructive criticism to help you to avoid them in the future.

CONTRIBUTION BY DR. FRITZ B. TALBOT

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THE RELATION OF DIET TO THE DEVELOPMENT OF CHILDREN, WITH SPECIAL REFERENCE TO THE TEETH[1]

THE diet of the growing child and the proper digestion of the food is the most important single factor in considering the physical welfare of children. It is of importance also in dentistry, in that upon the general well-being of the child depends the health of his teeth and their proper development. This paper will deal more with the problems belonging especially to pediatrics, but problems which directly or indirectly have a bearing on the health of the teeth. In order to understand the rôle of diet in infancy and childhood a few general principles must be outlined.

The food is composed of fat, carbohydrate, protein, salts, and water. The fats and carbohydrates supply fuel to the body with which to keep it warm; the protein builds up tissues, the salts build up the skeleton and teeth; while water carries away the waste products and dissolves the solids.

The period of infancy especially and early childhood is a period of rapid growth. There is a great increase of all the tissues during this time to make this growth possible. The quality and quantity of the food plays an important part in obtaining proper growth and, therefore, must be considered in detail. This will be done later in this communication.

The protein needs are at least 2 grams of protein per kilogram of body weight. This amount of protein must be given

[1] Paper submitted to the Academy of Dental Science, Boston, Massachusetts.

in the diet so that there will be enough to form new tissues in muscles and organs. The adult differs from the growing child in that the adult requires only enough protein to replace that used up in the wear and tear of every-day life, while the child must have enough to replace wear and tear, and also a surplus to deposit in the body to supply the necessary materials for normal growth.

The metabolism of the salts is comparable to that of protein, in that in childhood enough salts must be present both to replace those lost during wear and tear and also to build up new tissue. The present knowledge of the metabolism of salts is very limited, but a few facts are sufficiently well established to be outlined here.

The inorganic constitutents of the bones make up 40 per cent. of the dry residue and the organic constituents 60 per cent. "The inorganic constituents are chiefly calcium phosphate and carbonate, but there is also a little magnesium and a trace of fluorids and chlorid" (Matthews).

The teeth are composed of cement, dentin, and enamel. The cement has the same chemical composition as bone. Dentin, which makes up the greater part of the teeth, also has the same chemical composition, although its structure is different. The enamel is the hardest structure in the body, and contains only 5 per cent. of water. In the human it is composed of 91 per cent. calcium phosphate. Calcium phosphate, therefore, is the most important single element in both the bones and the teeth, and it may be assumed also that anything that affected the one would also affect the other. In studying the diet and its relation to the teeth, the rôle of calcium phosphate in metabolism must be understood. It must be considered from the point of view of the metabolism as a whole and of the metabolism in diseases of the bony structure, such as rickets.

The calcium requirements of the body can only be determined by metabolism experiments in which the calcium in the food, urine, and feces is determined. The figures available are not numerous, but in man 0.4 to 1 gram of calcium oxid a day is required in the food to maintain an equilibrium, while a total

amount of 0.7 gram of calcium oxid a day is supposed to be a minimum. An infant should absorb at least 0.13 gram of calcium oxid in the day, according to Orgler, otherwise it will develop rickets. The average amount taken by all infants is between 0.17 and 0.18 gram per day. This amount, when compared to the adult requirements, is very high. The body weight of an adult is at least ten times as great as that of an infant five months of age, whereas his calcium requirements are only four times as great. If the infant were as large as the adult and still growing, it would require 2 or more grams of calcium per day to supply its needs. The high calcium requirement in infancy is, of course, due to the demands to make new bony structure. Before birth and during the months after birth, while the infant is nursed at the breast, the demands of the child are satisfied by the mother. Her calcium requirements are, consequently, increased, and unless extra lime is given in the diet to make up for this, her teeth and skeleton may suffer. This should be borne in mind by the dentist under whose care a nursing mother may come. After weaning, the supply of calcium to the child is not automatically regulated, and depends upon the person who prescribes the formulas which the infant receives.

The period of infancy is that of greatest growth. The body doubles its birth weight in the first five months and triples it in the first year. During that period the source of the food is principally milk, while in childhood a great variety of foods are given, and growth is not so rapid. It is during infancy when the commonest disease of the bones—rickets—is found. The results of rickets are seen in childhood, but a child rarely develops rickets after the second year. Since rickets is the disease most often associated with delayed dentition, it must be considered here. Rickets is characterized by having a deficient supply of calcium in the skeleton. This deficiency in calcium is explained by:

1. Poor hygiene and lack of exercise.
2. Abnormality of the glandular secretion.
3. Calcium starvation, too little in the food.

4. Withdrawal of calcium from the body because of faulty digestion or metabolism.

The effect of the organs of internal secretion, such as hypophysis, thyroid, ovaries, and testes, on the metabolism of calcium is so little understood that at present judgment must be withheld. The burden of proof lies with those who believe that these organs as a whole or singly have any influence at all. For practical purposes at present, at least, it must be assumed that they have no effect on the calcium metabolism.

Although poor hygiene and lack of exercise is usually present in rickets, sufficiently accurate data has not been gathered to prove that it is the cause. The writer believes that it is coincident, and that the cause should be sought in the diet.

Calcium starvation, or too little calcium in the food, is also quite unusual in early childhood or infancy, when the diet is composed in large part of milk. But all children do not drink milk, and it is more than possible that the diet may contain so little milk and other foods containing calcium that there is actually calcium starvation. When this is found to be the case, the diet should be so regulated as to supply enough calcium in the food, although inorganic calcium in water and lime-water can be absorbed as well as organic calcium. It is, however, in such small amounts that it is of no practical value. According to Sherman, "A quart of milk contains rather more calcium than a quart of clear saturated lime-water, and by far the most practical means of insuring an abundance of calcium in the dietary is to use milk freely as a food."

Calcium starvation, with the resulting diminished calcium in the bones, has been produced artificially in animals. This has been accomplished by feeding animals food deficient in calcium, on an acid food, or a combination of the two. After feeding animals for a period on such a food, they have become clinically rachitic, with enlarged epiphyses. The microscopic appearance of their bones differs from that of true rickets. The bones have a very low calcium content, but have not lost rela-

tively as much magnesium as is lost in true rickets. The following example from Rolopf shows how the bones of a growing puppy on a calcium poor diet differs from one on a calcium rich diet:

Diet.	Dry substance.	Ash.	CaO.	Phosphoric acid.
Ca poor shoulder-blade.....	2.678	1.178	0.638	0.499
Ca rich shoulder-blade.....	9.021	5.240	2.861	2.211

Unfortunately, similar observations and analyses were not made of the teeth, so that it will have to be assumed that what applies to the bony skeleton also applies to the teeth. The calcium content of the different foods will be given later and the diet commented upon. It will then be quite clear that with certain combinations of food a diet deficient in calcium is quite possible even in the diet of older children. During infancy this will not be possible when the food is made up of human or cow's milk except under unusual conditions.

Some other agent must, therefore, be a causative factor of rickets in infancy and later life. If there is sufficient calcium in the food, enough ought to reach the bones and teeth for normal growth. This does not always follow. The metabolism of salts is very complicated, but there seems to be enough evidence to conclude that certain abnormalities of digestion may so affect the absorption of calcium that even with enough in the food, rickets develops. This may happen when as a result of some abnormality of digestion or metabolism, more calcium is lost in the urine and feces than is taken in the food. An excess of fat in the food, for instance, may draw more calcium out of the body than was ingested in the food (Orgler). Carbohydrate may affect the calcium retention beneficially when given in normal amounts, and harmfully when given in enough excess to cause indigestion.

The following table, taken from Sherman,[1] shows the calcium content of the common articles of food:

[1]From Sherman's Chemistry of Food and Nutrition, page 290.

APPROXIMATE AMOUNTS OF CaO IN FOOD MATERIALS

Food.	CaO per 100 grams edible substance.	CaO per 100 grams protein.	CaO per 3000 calories.
Beef, all lean	0.01	0.045	0.25
Eggs	0.09	0.66	1.7
Egg yolk	0.2	1.3	1.6
Milk	0.17	5.1	7.2
Wheat, entire grain	0.06	0.4	0.52
Patent flour	0.025	0.26	0.2
Low-grade flour	0.04	0.3	0.4
Rice, polished	0.01	0.1	0.08
Oatmeal	0.013	0.008	0.1
Beans, dried	0.22	1.0	1.9
Peas, dried	0.14	0.4	0.9
Beets	0.03	1.9	1.9
Carrots	0.08	7.6	5.2
Parsnips	0.09	5.3	4.1
Potatoes	0.02	0.9	0.7
Turnips	0.09	6.9	6.4
Apples	0.014	3.5	0.6
Bananas	0.01	0.7	0.3
Oranges	0.06	7.8	3.4
Pineapples	0.02	5.2	1.4
Prunes, dried	0.06	1.8	0.6
Almonds	0.30	1.4	1.4
Peanuts	0.10	0.4	0.5
Walnuts	0.11	0.6	0.5

This table shows that beef, polished rice, and bananas are extraordinarily low in calcium; that the more highly refined wheat is, the less calcium does it contain. Milk, oatmeal, and beans stand out as containing large amounts of calcium, and obviously should be given in large amounts when it is desirable to feed more calcium to the body.

Phosphorus is necessary as well as calcium to form skeleton and teeth. It is deposited in both structures in combination with calcium, but in considering the diet it has not been considered separately from calcium. If it were necessary to consider phosphorus separately, the following from Sherman sums up the essential points to remember: "In general, the most practical and economic method of securing an abundance of phosphorus in suitable forms is by free use of milk, eggs, vegetables, and such cereal products and bread-stuffs as contain at

least a part of the outer layers as well as the inner portion of the grains."

The diet must be so balanced, however, that besides containing sufficient calcium it must also have enough calories for growth and enough protein to supply the needs of the tissues. It must be given in amounts that will not tax the digestion and be adapted to the needs of the individual child.

The diet of the infant that is so unfortunate as to be artificially fed belongs in the specialty of pediatrics. In early childhood breakfast should be composed of fruit, cereal, milk, eggs, or bacon. Dinner of meat or broth, one starchy vegetable, such as potato or rice, and one green vegetable, such as string beans, spinach or peas, with a dessert containing milk. Plain cheese, containing all the calcium of milk, should be used much more often than is the custom in this country. Supper should be simple, and consist of cereal and milk, or bread and milk, with some cooked fruit as a dainty.

Care of the bowels, mastication, and other good habits of eating will ensure a good digestion. These, with a proper diet, should ensure normal secretions in the mouth. It is advisable that a very young child should have a piece of dry toast or cracker to chew when the teeth have come through the gums, so that it will learn at an early date the habit of thoroughly masticating its food. Dry toast, baked in the oven, is especially good for this purpose, as it is hard and requires chewing and is also easily digested. It is my habit to give an infant a piece of toast to chew whenever enough teeth have erupted to give two biting surfaces.

There are many infants who do not cut their first teeth until the latter end of the first year, despite the fact that their diet is well regulated, and they have no evidence of rickets. Inquiry will usually elicit the information in these cases that this is a family peculiarity. Conversely, some infants are born with teeth.

There are no drugs known that will affect the growth of teeth. Rickets is said to be affected favorably by teaspoonful doses of phosphorus and cod-liver oil in the proportion of 1 : 3000.

The diet in later childhood depends upon the digestive capacity of the child. The most common errors of diet are to give too much sugar, sweets and candy, and too much fat. Sugar with every article of food is not necessary because it is converted into sugar before absorption. No child should receive more than one teaspoonful of sugar a day. Candy is bad for most teeth and should be strictly limited. The earliest symptom of too much sugar in the food is loss of appetite; the complexion becomes pale and muddy, the flesh flabby, and the abdomen often becomes distended.

Fat, when given in excess, may cause indigestion, with bulky, pale stools. These stools are composed almost entirely of fat. It has been shown that fat has the power of combining with the salts, including calcium, to make soaps. Enough calcium may be thus drawn from the body to result in a "negative balance," viz., more calcium is lost than is taken in the food. Although the gross appearance of the stools usually tells when there is an excessive amount of fat in the diet, it can sometimes only be determined by examination under the microscope. Cod-liver oil should not be given to such cases. The milk must be skimmed and the butter limited.

It is not necessary to emphasize the fact that no matter how good the diet, the digestion will not be good without healthy teeth and proper mastication. Improper diet, with unhealthy teeth, makes a vicious circle which will require time and careful treatment to cure. There are no principles of biologic chemistry which the writer is able to find which will explain the formation of tartar deposits on the teeth, and one must fall back on the general principles of dietetics and hygiene both of the teeth and the body to prevent its formation. These principles are well known and are based on experience.

CLINIC OF DR. CHANNING FROTHINGHAM

HARVARD MEDICAL SCHOOL

AORTIC ANEURYSM

An Example of Case Teaching at The Harvard Medical School, with Report of Autopsy

IN selecting cases for the course in case teaching for the third-year students at the Harvard Medical School those cases are chosen, in so far as possible, in which autopsies have been performed, in order that the case may be completely presented. The entire case, including history, physical examination, and clinical pathology, is presented in detail from the hospital record, and the various possible diagnoses recorded as they suggest themselves during the presentation of the case. After all the evidence has been presented, a discussion of the findings is held in order to select from the various possibilities the proper diagnosis. This is then confirmed or not by reading the pathologist's report. The case is then reviewed to emphasize the important points, or to endeavor to explain why any mistakes or omissions in diagnosis were made.

The case presented at this exercise is that of a man fifty years old, who, for the past five years, had been doing clerical work, although formerly he had been a grocer and bartender. He entered the hospital complaining of cough and difficulty in breathing and swallowing. The family history showed that his father died of locomotor ataxia at the age of seventy-five. His mother, brother, and two sisters are living and well. He has lost no brothers or sisters. There is a history of tuberculosis in one paternal uncle and of carcinoma in a maternal uncle.

He was married and lived with his wife for seventeen years, but they have now separated because of alcoholism on the wife's part. There have been no children or miscarriages resulting from the marriage.

The past history shows that he had measles, chickenpox, whooping-cough, typhoid fever, and scarlet fever in childhood. At eighteen years he was operated upon for a paraphimosis. At twenty years he had an attack of gonorrhea and has had three subsequent attacks, the last one when he was forty years old.

Since he was forty he has been bothered with hemorrhoids, which have at times bled considerably. He has noticed a slight increasing deafness for the past few years.

He has drunk a good deal of alcohol in the past, but usually periodically. He would drink 2 or 3 pints of whisky a day for a week and then would not drink any alcohol again for several weeks.

The family and past history in this case should call one's attention to certain points. Thus, the family history suggests as possibilities syphilis, tuberculosis, and cancer, although only rather remotely. The age of seventy-five is pretty well advanced for death from locomotor ataxia, and so some doubt would naturally be raised as to whether the father really died from that form of syphilitic infection. From the past history, one should bear in mind gall-stones as resulting from the typhoid fever, and possibly injury to the vascular system, endocardium, or kidneys as a result of the scarlet fever. Again, syphilis should be borne in mind from the repeated attacks of gonorrhea, as we know that such an infection is often coincident with a urethritis, and often overlooked. The possibility of cirrhosis of the liver should be suggested from the alcoholic history and hemorrhoids.

With these possibilities recorded, we turned to the present illness of the case. This started about two and a half years before admission, with pain in the right shoulder. The pain would come on while he was walking, and be relieved by lying down, or by supporting his right arm and shoulder. Associated with the pain was some numbness in his left hand, and shortness

of breath preceding the attack. For the past two and a half years he has been short of breath upon exertion, and would have to sit up often at night to breathe. During the past year the pain has also been present at times in his left arm, and also in his chest. This pain was sharp in nature, and would come and go, sometimes lasting as long as an hour. Associated with the pain in the chest he would sometimes feel quite uneasy and become blue in color.

During the past two and a half years he has had a very pronounced cough and has spit up a considerable amount of sputum. At this time he was suspected of having tuberculosis and recommended for treatment at the state sanatorium. He never spit up any blood until two weeks ago, when he raised a few clots of blood, but does not feel sure that they were not vomited rather than expectorated. About one year ago he began to notice that his voice became hoarse on exertion or when out in the cold. This has gradually become worse. During the past six months he has felt faint often, and once actually fainted on the street car. For the past two months he has noticed difficulty in swallowing. He finally gave up work one month before entrance to this hospital, and has been in bed most of the time with the disturbance to breathing and swallowing becoming progressively worse. During the past year he has lost about 60 pounds, most of it lately. Except for symptoms mentioned above he has no complaints, and his other functions are apparently normal.

The history of the present illness in this case directs our attention to the chest and mediastinum, and we find signs suggesting some tumor formation in the upper chest cavity which obstructs breathing and swallowing and causes referred pain in the arms. The tumor mass may be a begnin tumor, an aneurysm, a malignant growth, or enlarged glands associated with some disease. The possibility of the heart or kidneys or blood-pressure being at fault to account for the shortness of breath, and of some infiltrative process in the lungs to account for the cough must be considered.

The physical examination showed a well-developed, but

poorly nourished man who was conscious and clear mentally. He sat up in bed and, at times, exhibited some difficulty in breathing. His skull, scalp, hair, face, ears, nose, and skin presented nothing abnormal. His pupils were equal, regular in outline, and reacted to both light and accommodation. His eye-grounds were normal in appearance. His breath was foul, his teeth showed cavities, and there was pyorrhea and redness about the gums. His tongue was slightly coated and showed a slight tremor. The tonsils and pharynx were normal. His voice was husky, but the vocal cords could not be seen because the epiglottis obstructed the view and seemed to be paralyzed, as it did not move. In the neck there were no abnormal pulsations nor any enlargement of the lymph-nodes or thyroid. The lymph-glands throughout the body were not enlarged. The thorax was asymmetric, in that the left side was flatter than the right. On the left side expansion with inspiration was absent. No abnormal pulsations were seen.

The heart apex was palpable in the eighth left interspace in the posterior axillary line 23 cm. from the midsternum. There was dulness 2 cm. to the right of the sternal edge in the third and fourth spaces. On account of surrounding flatness in the left chest the left heart border could not be percussed. No thrills were felt on palpation. The heart sounds were regular, rapid, and of fair quality. Except for a soft systolic murmur heard only at the apex there were no murmurs. The aortic second sound was somewhat greater than the pulmonic second sound.

The left radial pulse was entirely absent, nor could any pulsation be made out in the left brachial artery. The right radial pulse was regular and of fair quality. The vessel walls were slightly palpable. From the left arm superficial veins ran across the front of the chest on the left side toward the ensiform region, where a large vein was seen running down the abdominal wall until it disappeared at the umbilicus. The blood in the superficial veins ran from above downward.

The blood-pressure was 115 systolic and 75 diastolic.

On percussion over the lungs the entire left side of the chest

front and back was flat, except from the angle of the scapula to the base behind, while on the right side there was hyperresonance. The lower border of the resonance in the right back descended well with inspiration. On auscultation the right lung presented normal breath sounds and no râles. The lower left chest, front, back, and axilla, presented markedly diminished breath sounds without râles. The tactile fremitus was also diminished. In front, from the left apex to the third interspace, there was distinct bronchial breathing without râles and with bronchial voice sounds, and over a corresponding area in the back there was distant bronchovesicular breathing without râles. Over the upper front the tactile fremitus in the left was increased, while behind it was much diminished. The abdomen was negative and the liver, spleen, and kidneys were not palpable.

The hands show some wasting of the muscles and a slight tremor, the left arm was colder and slightly more cyanotic than the right, and the veins on this arm were more prominent. Sensation to rough tests was apparently normal and the reflexes were present and equal on both sides.

On entrance the temperature was 99° F., the pulse 104, and the respiration 25. The urine showed the slightest possible trace of albumin and a few hyaline and granular casts. Subsequent examinations showed no albumin. The blood examination showed 100 per cent. hemoglobin, white count 19,400, with 90 per cent. of polymorphonuclear neutrophils. The stools showed no parasites, eggs, pus, or blood. The blood Wassermann was double plus. The electrocardiogram showed left ventricular hypertrophy, but otherwise a normal complex. The Roentgen report stated that there was a large uniformly dense shadow obscuring the entire lung field on the left and the outlines of the heart and aorta, except for a small bit of air-filled lung in the lateral aspect of the chest just above the diaphragm. There was no heart shadow to the right of the sternum and the trachea was displaced anteriorly. The findings suggested to the roentgenologist the presence of a neoplasm in the mediastinum. The mass which causes this shadow was rounded and smooth, but did not pulsate.

Of the possibilities suggested by the patient's history, it is possible now to rule out as a result of the physical examination and laboratory studies certain of them. Thus there is nothing to point to cirrhosis of the liver, as the liver is not palpable and there is no evidence of obstruction to the portal circulation. In fact, the enlarged vein in the abdominal wall suggests that the blood can reach the heart more easily from below. There is no evidence of any injury to the vascular system, endocardium, or kidneys referable to the scarlet fever, as the vessels are not palpably sclerosed, there are no important murmurs in the heart, the blood-pressure is not elevated, nor is there any evidence of nephritis. Against tuberculosis is the fact that although the signs in the left chest are extensive, there are no râles or evidence of pleural fluid. It would seem almost certain that so extensive a tuberculosis would be accompanied by râles. The location of the heart apex is doubtless due to the displacement of the heart, as there is no increased blood-pressure or valvular lesions to cause it to hypertrophy. Also the *x*-ray does not show the right border to the right of sternum as percussion suggested, so it presumably is not tremendously enlarged. Enlarged glands due to blood disorders are ruled out by the blood examination, and the limitation of the enlargement to this one area would be unusual in Hodgkin's disease.

Of the provisional diagnoses made, there remained, therefore, syphilis, which was confirmed by the Wassermann test, benign tumor, cyst, or malignant disease of the mediastinum, and gall-stones, which, of course, may be present, but which may be ruled out as being a factor of importance in the case. If syphilis is the cause of the signs and symptoms, it must be one of its end-results as an aneurysm, resulting from invasion and destruction of the medial count of the aorta by the spirochetes than a gumma, as the tumor mass is too large for that. Against aneurysm is the absence of pulsation on palpation or *x*-ray study, and the absence of tracheal tug and pupillary changes. In favor of aneurysm over benign or malignant tumor is the fact that it is more common than primary tumor in this region, and that a syphilitic infection is known to exist. The elevated tem-

perature and leukocytosis is more consistent with a tumor, especially a malignant one, than with an aneurysm. The disturbance to swallowing and breathing and the disturbance to the return of blood to the heart from above, as shown by the collateral circulation on the abdominal wall, simply mean pressure in the mediastinum upon the bronchi, esophagus, and superior vena cava or some other large vein, and do not help in differentiating between aneurysm and tumor. The emaciation does not help in the differential diagnosis, as it was obviously due to starvation dependent upon difficulty in swallowing. The notes in the record by the various doctors who examined the patient showed that difference of opinion existed among them in regard to the nature of the tumor mass.

The subsequent course of the disease showed that the patient was running a temperature which ranged from normal to 102.5° F. and a pulse which ranged from 85 to 120 per minute. The respirations varied between 20 and 30 per minute. The outstanding symptoms were pain in chest, dyspnea, and troublesome cough, from which only a thin watery sputum was raised. These symptoms required opiates frequently, and the difficulty in breathing became steadily more pronounced, until the patient practically choked to death three weeks after admission. During this period the leukocyte count continued elevated, but no other signs suggestive of any complicating disease occurred. Observation during these three weeks failed to add any further information to aid in differentiating between tumor and aneurysm.

The anatomic diagnoses made at autopsy were: aneurysm of the arch of the aorta, bronchopneumonia, gangrene of the lungs; posterior mediastinal abscess communicating with the esophagus, and fibrinopurulent pleurisy.

The pleurisy and bronchopneumonia were probably terminal affairs and did not exist at the time of admission. They probably contributed to the continued temperature and leukocytosis, but were not the original cause of it. The abscess in the posterior mediastinum probably was of slightly longer duration, and also helped to account for the leukocytosis and fever. The origin

of the leukocytosis and fever probably lay in the necrotic tissue caused by pressure from the aneurysm mass.

A clot was found extending into the left subclavian artery, which accounted for the absence of radial pulse on the left. Also in the aneurysmal sac there was much old clot, which probably accounted for the absence of pulsation in the fluoroscopic examination. The heart was apparently just crowded out of its normal position by the tumor mass, as was thought before death.

This case demonstrates the disturbance caused by a large tumor mass in the mediastinum, and reminds one that the usual signs, such as tracheal tug, pulsations, and bulging of the chest and irregularities of the pupils, may be absent in the presence of an aneurysm.

One should emphasize the fact that pain without apparent cause in the arms or chest or persistent cough without pulmonary involvement should always make one suspicious of the starting of trouble of a serious nature in the mediastinum.

CLINIC OF DR. GEORGE R. MINOT

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FOUR CASES WITH ENLARGED SPLEENS

Case I.—Banti's Disease. Discussion of Term "Splenic Anemia." The Symptoms, Blood-picture, and Differential Diagnosis of Banti's Disease. Splenectomy in Banti's Disease and Other Conditions.

Case II.—An Instance of Banti's Disease Mistaken for Peptic Ulcer. A Further Consideration of the Differential Diagnosis of Banti's Disease.

Case III.—Myelogenous Leukemia with a Low White Count. Illustration of the Value of Critical Blood Examination.

Case IV.—Typical Chronic Myelogenous Leukemia. Comments on Symptoms and Treatment by Radium.

I AM going to show you today four cases that have enlarged spleens, and I shall discuss their differential diagnosis and treatment.

CASE I

The first case is that of a boy aged fourteen, who gives the following summarized history:

Family history and *social history* entirely negative.

Habits good.

Past History.—He has never been "very strong" all his life. Tonsillectomy was performed when he was three years old. He had mumps at the age of five; measles at the age of seven; and pneumonia at the age of nine.

Present Illness.—When ten years old, now four years ago, he began having "indigestion attacks." These attacks consisted of a dull generalized abdominal pain, associated with some distention and belching of gas, lasting for a few days to two weeks. Sometimes accompanying this disturbance there would be nausea, rarely vomiting, and not infrequently diarrhea for two

or three days, with three to five stools a day. No blood was seen in the vomitus.

These attacks have persisted and increased in severity and frequency. At first they occurred every five or six weeks, but in the past year they have come at least every two weeks. During the past there has also been some gas and epigastric discomfort more or less continuously, with a poor appetite. There has been no sharp pain, no known fever, and these gastro-intestinal symptoms have rarely caused him to want to stay in bed.

During the past three years he has felt constantly tired and has grown increasingly weaker. Three years ago he weighed 59 pounds; a year ago, 62; and now, 59. Thus he is now much thinner than three years ago because he has grown several inches during this time.

Ten months ago he noticed his abdomen was abnormally large, and he thinks it has grown considerably bigger since then. His relatives state that he has been more or less pale for three years, and that he has been distinctly pale for a year, being paler now than a year ago.

During the past six months he has slept poorly, has had occasional headaches, and has been slightly short of breath.

Physical Examination.—Physical examination shows a pale, poorly nourished boy, with a large abdomen, and a few small purpuric spots on his legs. His temperature has been about 100° F. each evening for the past four days (since he has been under observation).

It is to be noted that it is not unusual for cases of his condition to have a degree or so of fever.

The pupils, throat, and mouth are negative. No lymph-nodes are palpable except for a few small pea-sized ones in the submaxillary and cervical regions.

The lungs are negative, and so is the heart, except for a slight functional systolic murmur.

The abdomen, besides appearing large and rather distended, shows slight tenderness in the epigastrium. The spleen is very much enlarged, reaching the midline at the umbilicus and ex-

tending 4 cm. below it in the left nipple line. It is very hard and firm, but not tender. The liver edge is easily felt 4 cm. below the costal margin, and is firm and non-tender. The upper border of liver dulness is at the sixth rib.

There is no jaundice and there are no dilated veins. There is no evidence of shifting dulness in flanks to suggest ascites. Neither kidney is felt. The genitals and reflexes are normal. The extremities are negative except for a slight clubbing of the fingers.

Though it is not usual to see clubbing of the fingers in the disease which this patient has, it may occur.

Laboratory Findings.—Urine and stool examination negative. Wassermann reaction negative.

Blood Examination.—Hemoglobin, 55 per cent.; red count, 3,300,000.

The *red cells* are achromic and show a definite abnormal variation in size, which is not marked. There are no very small red cells and no very large ones. There is only slight variation in shape. Rarely a polychromatophilic cell is seen. No stipling, Howell-Jolly bodies or blasts are present.

The *white count is 4000.* The differential count:

Polynuclear neutrophils	53 per cent.
Small lymphocytes	38 "
Large lymphocytes	3 "
Large mononuclears	6 "

No eosinophils or mast cells are seen.

Platelets are definitely considerably diminished in numbers.

Other blood tests revealed no positive information of value.

This case is a typical instance of *splenic anemia* in the stage of transition to the condition classically known as *Banti's disease.*

The term *splenic anemia* frequently has been used to include several distinct types of conditions, and its use should be restricted. Many types of conditions with enlarged spleens and anemia have been in the past called splenic anemia; I will refer to these later. The term *splenic anemia* is best reserved for the early stage of what is known as *Banti's disease;* or it may be bet-

ter to discard the term entirely and refer to such cases simply as Banti's disease in an early stage. The etiology and pathogenesis of this disease remains obscure. It usually occurs in young adults and runs a chronic course, being rather more common in men than in women. The disease is classically divided into three stages, which, of course, merge one into another, and are not in any given case sharply differentiated. Primary enlargement of the spleen is the first recognizable sign of the condition, and represents what is known as the first stage of the disease. This alone may be present for years. The second stage is associated with progressive enlargement of the spleen and anemia. The spleen is usually greatly enlarged, commonly reaching the umbilicus, and not infrequently being larger. As the anemia becomes evident, symptoms referable to it and to the enlargement of the spleen occur, such as increasing weakness, pallor, digestive disturbances, and abdominal discomfort; all of which symptoms are exhibited by this patient before you. It is to these first two stages of Banti's disease that the term "splenic anemia" is best applied.

As the disease progresses the third stage of this symptom complex, classically known as *Banti's disease*, occurs. In this stage the individual shows signs of cirrhosis of the liver and a failing portal dirculation, as evidenced especially by ascites.

The disease usually runs an extremely chronic course over a period of many years, and in some instances may never progress to the third stage; or it may rarely run a much more rapid course, so that the typical Banti's disease picture may be exhibited within perhaps a year. Though the disease is usually slowly progressive, there may be periods of remissions in the symptoms and the anemia. Such remissions are rarely very marked, and rarely if ever as marked as the marked remissions that may occur in pernicious anemia.

An important and frequent symptom that these cases often exhibit is hemorrhage from the gastro-intestinal tract. This is probably in many cases due to mechanical causes dependent upon the enlarged spleen, and in some instances from the cirrhosis of the liver. The bleeding may occur from gastric erosion

because of congestion, or from rupture of esophageal varices. Hemorrhages from other sources are not uncommon in these cases, particularly from the nose, as has occurred in the case before you. Such a hemorrhage often occurs without any clearly recognized defect of the blood factors associated with pathologic hemorrhage, and often without any recognized local mechanical defects.

Some cases, usually in the more advanced stages, do show a definite clear-cut hemorrhagic diathesis; a secondary purpura hemorrhagica. This is evidenced especially by spontaneous hemorrhages from the gums and nasal regions, and purpura, associated with marked diminution of the blood-platelets. The purpura on the legs of this boy is probably associated with distinct diminution of platelets in his blood.

Another factor that may be responsible for some of the pathologic hemorrhages is defective liver function. In some instances probably pathologic blood defect helps to exaggerate the gastro-intestinal hemorrhages largely dependent upon mechanical causes.

The defects of the hemopoietic organs in this disease would appear to be particularly *impaired blood formation*, but of a relatively normal type in contrast to that of an abnormal type seen in pernicious anemia. There also appears to be present sometimes some increased hemolytic activity. This is never marked.

The evidence of impaired blood formation is reflected in the peripheral blood-picture by a leukopenia, with not infrequently a relative lymphocytosis and absolute diminution of the polynuclear cells; diminished numbers of platelets, and red cells of a type seen in so-called chronic secondary anemia, with little evidence of their active regeneration. Leukopenia is a feature of the condition. The white count in uncomplicated cases when the disease is well established is probably never over 7000; usually about 3500. Following a severe hemorrhage the count may rise, but proportionally not so much as after a similar amount of hemorrhage from accident in a healthy person. Similarly, other complications may temporarily increase the

count. The red-cell picture in these cases, in the absence of complications, is as stated: that of chronic secondary anemia with a relatively lower hemoglobin than red-cell count; rarely do the cells vary greatly in size or shape; and though polychromatophilia and stipling may occur, they are practically never marked, nor are the reticulated red cells ever increased more than slightly. Apparently, relatively early in the disease, even though the spleen may reach the umbilicus, there may be evidence of a more active marrow, so there will not be a leukopenia, and the white count may rarely be as high as 10,000. Under such conditions the polynuclears may form a relatively high percentage (70 per cent.) of the white cells, and the "large mononuclear" cells are often increased. It is perhaps not uncommon to see the "large mononuclear" cells increased even with the lower white counts, and sometimes they appear in character somewhat different from those usually seen in normal bloods. With the higher counts an occasional polynuclear eosinophil or mast cell may be found; these are seldom found with the lower counts. Under such circumstances the red cells may show relatively more signs of youth, and the platelets, too, may be slightly increased, and not infrequently large in size.

The evidence of hemolytic activity, increased blood destruction, is particularly seen in increased excretion of urobilin and by increases of bile pigments in the plasma. This apparently is never marked, and does not occur early, and not infrequently is absent. The increase of bile pigments in the plasma is at times undoubtedly dependent upon the liver cirrhosis. Definite jaundice is never marked and is rare in the disease, probably not being seen until cirrhosis develops.

It is quite probable that the spleen normally has a regulatory action over the marrow, and it would seem that in this disease the altered splenic function was of such a nature that it caused an abnormal depression of the marrow without any marked alteration of its character. Perhaps early in the disease, or at times of remission, the marrow is able to overcome this depression. This impaired blood formation is probably the chief hemopoietic defect. As increased red-cell destruction is not

marked, it seems it must play a relatively small rôle in the production of the anemia.

Differential Diagnosis.—Among the conditions that in the past have been called splenic anemia, and which are to be distinguished from Banti's disease, are Gaucher's disease, von Jaksch's anemia, and the various types of hemolytic jaundice.

Gaucher's disease is characterized pathologically by a peculiar large vesicular cell hyperplasia of the spleen, while the spleen of Banti's disease shows chiefly simply increase of fibrous tissue. Gaucher's disease is much rarer than Banti's disease. The blood-picture may apparently be the same as in Banti's disease. The condition often is not recognized in life. The following points are unlike what occurs in Banti's disease and help to distinguish Gaucher's clinically. It begins insidiously in childhood; a family history is often elicited; the spleen reaches enormous proportions; and the liver is often large.

The condition known as *von Jaksch's anemia*, or anemia infantum pseudoleukemia, is a symptom complex that occurs in infants and children and not in adults. This has been well discussed by Stillman in The American Journal of The Medical Sciences (1917, vol. cliii, 218). It is characterized by marked enlargement of the spleen, with slight enlargement of the liver, and sometimes with enlargement of the superficial lymph-nodes. The blood-picture shows an anemia with a low color index, and a persistent leukocytosis of varying degree. The platelets are increased. Nucleated red cells are nearly always present, frequently in large numbers, and the red cells often show a lot of polychromatophilia and stipling. These features of the red-cell picture are in contrast to that of Banti's disease, as are the white-cell and platelet pictures. This type of anemia is very frequently associated with rickets, and sometimes with syphilis, although it may occur without association with either of these diseases. Exactly what relation the symptom complex has to rickets or syphilis has not been definitely decided. It has been thought by some that this condition is simply the form of Banti's disease that occurs in the infant. However, it seems more likely that it is not. Cases of von Jaksch's anemia tend to

recover; in contrast to cases of Banti's disease, which tend to progress.

The acquired and familiar forms of *hemolytic jaundice*, characterized by constant increased blood formation in the presence of constant increased blood destruction, with enlargement of the spleen, may present many variations from their typical picture. Such variations may be dependent upon fluctuations of the anemia and the degree of activity of the hemolytic organs, and the progressiveness of any given case. These cases have been called to our attention particularly in recent years, and in the past have not infrequently been called splenic anemia. The severest cases of the acquired type appear to approach in character the more chronic hemolytic type of pernicious anemia cases with enlarged spleens. I shall mention some points by which hemolytic jaundice may be distinguished from Banti's disease. A family history may be obtained in the familial form; the individuals in this group are not so sick as in the acquired form; in fact, some cases remain for life in fairly good health; and, as Chauffard has said, the familial cases are more icteric than sick. Cases of hemolytic jaundice are nearly always more chronic, not infrequently much more chronic, than Banti's disease. Definite jaundice without bile in the urine is nearly always to be seen, while it is relatively rare in Banti's disease. This jaundice is dependent upon a marked increased blood destruction. The spleen on the average is not so large as in cases of Banti's disease. The histologic blood-picture of cases of hemolytic jaundice shows evidence of increased marrow activity which is often marked, in contrast to the decreased marrow activity usually seen in Banti's disease; one of the features of hemolytic jaundice being increases, often marked increases, of the reticulated red cells. Another feature is the typical diminished resistance of the red cells to various strengths of salt solutions, which is not present in Banti's disease.

Among other conditions not to be confused with *Banti's disease* are cases of pernicious anemia with enlarged spleens, certain ill-defined types of hemolytic anemia, the leukemias, polycythemia vera, various types of cirrhosis of the liver, splenic

enlargements associated with sepsis, malignant endocarditis, malaria, kala-azar, etc., as well as Hodgkins' disease and its allied conditions with enlarged lymph-nodes. Two other conditions, different and at times impossible to differentiate from Banti's disease, are thrombosis of the portal and splenic veins and syphilitic splenomegaly. It will not be possible to discuss all these conditions, and I will simply briefly refer to the last two.

Thrombosis of the splenic vein may give the same symptomatology and apparently the same blood-picture as Banti's disease. It is often impossible to differentiate splenic enlargement secondary to thrombophlebitis from that due to Banti's disease. Also it is to be noted that probably in some cases of true Banti's disease thrombosis of splenic, portal, and mesenteric veins may occur secondary to it.

Syphilis may cause anemia with non-gummatous, diffuse hypertrophy of the spleen, and much more rarely gummata in the spleen. In fact, it may be only because the Wassermann reaction is definitely and strongly positive that such instances can be distinguished from Banti's disease. Perhaps some such cases are simply instances of Banti's disease occurring in a patient who has had syphilis. One can always speculate in such instances on what relation the syphilis has to the splenic enlargement. It does, however, seem quite proper to separate from the other forms of splenomegaly those cases with large spleens in which a syphilitic cirrhosis of the liver exists, and where gummata of the liver are present. It is quite possible that such cases may show a rather different blood-picture than true Banti's disease, but this has not as yet been clearly demonstrated.

Treatment.—The only method of real therapeutic value in Banti's disease is *splenectomy*. Temporary improvement may be obtained by purely medical methods, such as proper feeding, rest, fresh air, iron, etc. Roentgen rays or radium in some instances—probably particularly in early cases—may cause some decrease in the size of the spleen and permit temporary improvement. In many cases it is without significant effect. This procedure may be tried before surgery, with a view to allowing the patient to be in the best possible condition for operation.

The question of such a procedure causing adhesions and making the operation more difficult has been discussed in the literature, and it would seem that it probably did not cause any serious adhesions in such cases. When the diagnosis is definitely established, splenectomy should not be done too hastily. Simple measures, besides radiation, perhaps, should be first undertaken, in order to have the patient in the best possible condition, and the most favorable time should be selected for the operation. Transfusion may be indicated in certain cases to improve the patient's condition before operation. In all cases before operation it is desirable to have an appropriately selected donor at hand, so that transfusion can be done after the operation if necessary.

In those early cases where the spleen is not very large and where the disease is apparently rather stationary and the patient in very fair health, it is most difficult to say that splenectomy should be urged, because some such cases may remain for years quite well. However, with any evidence of progress of the disease, splenectomy should be urged. Probably it would be wise to urge removal of the spleen in all cases where the diagnosis is definitely made after careful study of the case.

The earlier in the disease the operation is done, the better the results, and the more lasting the improvement of the symptoms. In many cases in the first and early second stage operation amounts to a complete cure. When the third stage is reached the operation is by no means contraindicated, but the chances of improvement are greatly lessened. The operation in this stage is also more dangerous. The operative mortality in the recent essentially unselected cases of Banti's disease is in the vicinity of 12 per cent. With selected cases and early cases the mortality percentage is apparently considerably reduced. Some cases operated when ascites was present have improved markedly; such cases, however, are apt to die within a few years. Though many of the cases operated upon in a relatively early stage of the disease appear to be completely cured, others may live for many years, feeling quite well, and then suffer a relapse, the postmortem examination showing changes asso-

ciated with cirrhosis of the liver and progress of the disease process.

Splenectomy is often a desirable therapeutic measure *in* some of the *conditions* that may be *mistaken for Banti's disease.* It may be advised in Gaucher's disease, but in such cases its benefit is probably never as permanent as in Banti's disease, and not infrequently gains little for the patient. In hemolytic jaundice in the acquired or familial form splenectomy is to be advised provided the patient's symptoms are bothersome enough to handicap his health, or there is evidence that the condition is progressive. In those mild familial cases where the patient has nothing to complain of except his appearance, it does not seem wise to advise operation. The results in hemolytic jaundice conditions are usually very successful; marked improvement is the rule, but apparently cure is not universal. Relapses may occur after splenectomy. If it were not for the risk of operation, probably all cases should be splenectomized as soon as the diagnosis is made; and undoubtedly the earlier the operation, the better the results. However, it must be remembered that in spite of the very brilliant results the exact cause of the condition is unknown, and although the spleen appears an important pathologic element in the disease, probably the true disease process does not reside wholly in the spleen, as is probably also true in Banti's disease.

Splenectomy in von Jaksch's anemia has seldom been done, and it seems that it should be reserved only for cases resistant to prolonged careful medical treatment, for such treatment often yields satisfactory results.

In pernicious anemia, splenectomy is at the best only a palliative measure, and probably is not to be advised in the vast majority of cases. It seems to be the means for inducing as good a remission as possible, but the gain to the patient is often slight when compared to the discomforts and risk of the operation. In selected cases it may be advised, if not urged. The cases that do best seem to be those with enlarged spleens without acute onset and with evidence of a good deal of hemolysis with an active marrow.

In typical myelogenous leukemia splenectomy is never to be advised; in fact, it is distinctly contraindicated, as it usually results in death. Recently at the Mayo Clinic, following radium therapy with shrinkage of the spleen, splenectomy has been practised, but the results would seem to show that though the mortality from operation is vastly lower than in untreated cases, the ultimate result gains nothing more for the patient than radium alone. It is possible that in certain rare cases of chronic myelogenous leukemia with a relatively small spleen and a low white count splenectomy may be done to advantage. However, reports on such cases are meager, and we do not really know how much is to be gained in such cases by this procedure over that which may be accomplished by radiation of the spleen. It is, I think, rather doubtful if splenectomy in such cases will do more for the patient than frequent, carefully given treatments with radium.

True syphilitic splenomegaly with secondary anemia is often resistant to antisyphilitic treatment, and in such cases splenectomy has been advised by Giffin, and seems justifiable and desirable when other forms of treatment have failed.

In other forms of splenomegaly than those just discussed splenectomy should seldom be done, and if so, only in certain unusual and atypical cases.

CASE II

The second case that I have to show you illustrates an instance where hematemesis was thought to be due to peptic ulcer, and where, in fact, it was, in all probability, dependent upon mechanical vascular disturbance from an enlarged spleen in a case of Banti's disease.

The patient is a man of thirty-four, who has been a New England farmer all his life. His history is as follows:

Family history, *social history*, and *occupational history* are wholly negative.

Past History.—As a child he had measles and whooping-cough, and rarely a mild sore throat. Ten years ago he had a septic hand. There is no history of any symptoms referable to

any of the systems of the body until nine years ago. There is no history of malaria, venereal history, or typhoid.

Habits.—Rarely has he taken any alcoholic drinks. He has never smoked or chewed tobacco. His living conditions have been good, and he has eaten in no abnormal manner.

Present Illness.—(As the history is a lone one, I shall not give every detail the patient described, but will try to point out the salient facts.) Nine years ago he began having indefinite abdominal discomfort associated with the belching of a good deal of gas. This discomfort did not occur at any regular intervals in the day, but he thought he belched more gas in the mid-afternoon than at other times. After some weeks he began having typical "heartburn," beginning usually two hours after eating and lasting for about an hour. There was no vomiting, rarely nausea. After some months of these symptoms he developed suddenly, one night, sharp pain, which he now says occurred in the epigastrium. He recalls no details about this attack, but was operated upon for appendicitis, and was later told that the appendix was found "pretty normal." Following the operation his abdominal symptoms subsided for some weeks, but then recurred. They became even more marked than before, and not infrequently he had sharp attacks of cramp-like abdominal pain of hours' duration on the right side of his abdomen, usually associated with vomiting, but occurring without relation to food or posture. These attacks continued until about a year after his appendix operation, when he consulted different doctors than formerly. They advised operation for "adhesions." This was done, and he says adhesions were found. Following this, he had no return of the right-sided abdominal pain, but the heart-burn occasionally recurred, and general abdominal discomfort continued.

For the next two years the symptoms fluctuated in periods of weeks, during which time he found he had fewer symptoms if he did not eat coarse foods or fried foods. Then, instead of having general abdominal discomfort, he began to have epigastric distress and a dull epigastric pain, particularly two hours after eating; this was associated with increase of "heartburn"

and gas. After some months of this, or now about five years ago, one day he suddenly vomited at least a pint of blood. In the next eight months his symptoms of "heartburn," gas, and epigastric distress continued, but he considers he had no more real epigastric pain. During this period he vomited blood at least five times. His friends state that he had been growing pale for a year before his first hemorrhage, and that he has been more or less pale ever since then. A diagnosis of peptic ulcer was made by his doctor at this time, and a gastro-enterostomy was done. He says he was told that two very small superficial erosions were found on the stomach. At this time he first knew that his spleen was enlarged, and he claims he was told that it had enlarged because it was nature's way to stop his hemorrhages. The facts are, of course, that its enlargement was responsible for his hemorrhage.

In the next two and a half years he had no hemorrhages, but the hyperacidity symptoms, gas, and epigastric discomfort continued, with rarely nausea. These symptoms perhaps were not so marked as in the eight months preceding the gastro-enterostomy. Following this operation, his color definitely improved somewhat, but he remained rather pale. During these two years he had a few slight nose-bleeds; and in the past three months he has had several more.

Six months ago, at a time when he was having relatively mild stomach symptoms, he vomited about a quart of blood. Since then he has vomited only six times, but each time there was about half a pint of blood. During these past six months epigastric discomfort has been prominent, occurring more or less persistently.

During the past nine years his bowels have been fairly regular except in the past six months, when they have been distinctly constipated. This may be dependent upon the fact that he has taken a lot of iron during this time. Hemorrhoids have been present for at least five years. In the past six months he has had numerous symptoms, evidently dependent upon his anemia *per se;* such as weakness, dizziness, slight swelling of his ankles, and palpitation. He has lost about 25 pounds in

weight during the past six months, so that he now weighs 105 pounds. Ten years ago he weighed 150 pounds, and fluctuated between 140 and 125 in the first eight years of his illness. This recent loss of weight can be accounted for by the fact that he has eaten very little food in recent months because of being allowed but little on account of the hematemesis. He does not know whether his spleen has grown bigger or not. He is inclined to believe it is the same size now that it was four and a half years ago, when it was first called to his attention.

Physical Examination.—The patient is thin and pale—a white pallor and not at all sallow or yellowish, as is so commonly seen in cases with some form of hemolytic anemia. His temperature has been normal during the three days that he has been in the hospital.

The pupils react normally. Tongue, throat, tonsils, and teeth are negative. No lymph-nodes are palpable except for some tiny ones in the neck. The lungs are absolutely negative.

The heart is negative except for a soft but rather loud systolic murmur heard over the pericardium, but loudest at the base. This is, of course, a functional murmur dependent upon the anemia. The pulse rate is 80. The blood pressure is: systolic 110; diastolic 75.

Examination of the abdomen shows that there is a fulness in the left upper quadrant. No superficial veins are to be seen that are definitely abnormally prominent. Palpation of the abdomen reveals definite tenderness in the epigastrium, but not elsewhere. There is no distention and no evidence of ascites. The spleen is very easily felt and extends, on deep inspiration, to just above the level of the umbilicus. At first one is inclined to believe the spleen is not very large, but upon palpation you will note that though it does not come within 6 cm. of the midline, it fills the left flank, and percussion shows it to extend under the ribs even higher up and further back than normal. It is a pretty big spleen. It is very firm and hard, and the notch is easily felt. The liver edge can just be felt on deep inspiration. Neither kidney is felt, and no other tumors are felt.

Rectal examination reveals some external and internal hem-

orrhoids. The genitals and reflexes are negative. The extremities are negative except for a very slight edema of the ankles.

Two *Roentgen rays* of the gastro-intestinal tract have revealed no abnormality except the gastro-enterostomy stoma. Roentgen ray examination of the kidney and gall-bladder regions was negative.

Laboratory Examinations.—Two stool examinations have been negative. Two urine examinations are absolutely negative except for the slightest possible trace of albumin.

The Wassermann reaction of the blood is negative.

The *blood examination* is as follows:

Hemoglobin, 45 per cent. Red count, 2,800,000.

The *red cells* are distinctly but not markedly achromic. They vary considerably in size, but no true microcytes are present. There are no true macrocytes. Variation in shape occurs, but is not so marked as variation in size, and there are no very bizarre-shaped cells. Polychromatophilia rarely occurs. There is no stipling and no Howell-Jolly bodies, or blasts occur. The reticulated red cells are 1 per cent.

The fragility of the red cells shows that hemolysis begins in 0.42 per cent. salt solution, is marked in 0.36 per cent., and complete in 0.28 per cent. This is within the normal limits, but one might expect in an individual who has had so much hemorrhage that the hemolysis would be complete in a weaker percentage of salt solution.

The *white count is 2300.* The differential count of 200 cells is as follows:

Polynuclear neutrophils	51	per cent.
Small lymphocytes	44	"
Large lymphocytes	1	"
Large mononuclears	4	"
Eosinophils	0	"
Mast cells	0	"

The *platelets* are definitely somewhat diminished.

The bleeding time is not prolonged. Coagulation time is normal and the clot retracts. The serum shows no evidence of containing abnormal amounts of bile pigments.

The presence of the enlarged spleen with leukopenia *in the presence of anemia taken together with the repeated hematemesis and the nine-year history makes, without doubt, the diagnosis of Banti's disease.* It is surprising to note that the patient states that he has never had his blood examined until now, although he recalls seeing twelve different doctors in nine years. Of course, any one doing good medical work should study the blood in any case with a large spleen or one showing pallor. The case further serves to point out the importance of studying all cases very carefully before operations are performed. It is probable that five years ago, when the spleen was known to be enlarged, the blood would have shown essentially the same features as now. Directly after a hemorrhage there may have been a leukocytosis, so that a count at such a time would not have been so valuable as counts some ten days or so afterward. In the chronic anemia resulting from hemorrhage, as in simple duodenal ulcer, the white count will remain somewhat above normal, so that the presence of leukopenia in such a case—as in the one I have just described—would be alone very suggestive of splenic anemia rather than only peptic ulcer. It is true, of course, that the symptoms the patient had suggest peptic ulcer, and that two small gastric erosions were said to be found at operation. However, such lesions and symptoms highly probably resulted from the splenic condition, and treatment should have been directed not at the result, but at the cause; by which I mean the spleen should have been removed, and not a gastro-enterostomy done. If this had been done, the patient probably would be vastly better than he is now. It is most unlikely that all the hemorrhages this patient had came from small gastric erosions. The greater amount of blood highly probably came from ruptured esophageal varices. Not only does the nature of the case suggest this, but the negative gastro-intestinal Roentgen ray examination favors this assumption.

The mistake made in diagnosis which this case illustrates has been made before by excellent men, and reports of other such cases are to be found in the literature. I have seen the mistake made several times. In the other cases the spleen was

not quite so large; and in one it was unusually difficult to feel, yet after its removal it was found to be fully three times normal size.

Individuals who have enlarged spleens from malaria, etc., occurring in the past, at the time they are seen, of course, may have peptic ulcer, and such cases may be confused with Banti's disease. It is in cases with the smaller spleens that the diagnosis is difficult; and in such instances one has to rule out not particularly peptic ulcer, but ordinary *atrophic cirrhosis of the liver*, because this condition often gives slight or moderate enlargement of the spleen and recurring hematemesis from esophageal varices. The salient points to differentiate between these two conditions consist in obtaining a history of alcoholism and evidence of late and not early splenic enlargement, which favors cirrhosis and not Banti's disease. A large spleen will always be much in favor of Banti's disease. With *Banti's disease* there usually will be a longer history of anemia than in cirrhosis, and in this latter condition there is not much anemia except in the terminal stage or after profuse hemorrhage. The blood-picture may help to differentiate the two conditions, but cirrhosis of the liver in its later stages may show a leukopenia. At times it may be impossible to distinguish these two conditions, particularly when they are in the advanced stages.

About two weeks after removal of the spleen in this case (Case II) the patient died from mesenteric thrombosis. Autopsy showed most extensive varices of the esophagus. There was a relatively small amount of cirrhotic change in the liver, and no ulcers or scars were found in the stomach.

CASE III

The third case that I have to present is an instance of where the diagnosis is established by careful examination of the blood. I will present the case briefly to you.

The patient is a man of forty years, who has always done clerical work.

The *family history* is unimportant.

His *habits* are good.

The *past history* is negative except for the fact that he has had migraine all his life.

The *present illness* is of about a year's duration, though he doesn't consider that he has been really ill for more than two months. A year ago he first began to have "dyspepsia," which has persisted and increased up to the present time, particularly in the past two months. This has consisted of frequently belching gas, a dull indefinite mild ache in the epigastrium, occasional nausea, and heart-burn. There has never been sharp pain or vomiting. During this time the bowels have been constipated. Four months ago he began having a dragging pain in the region of the left flank, accompanied by a dull ache in the small of his back. These sensations have been aggravated by increase of gas and by exercise. They have never been present when he lies down.

In the last six months he has become fatigued unusually easily, and he has in general felt weak and used up, even though he went on a long vacation two months ago. This sense of not feeling well and of being weak and tired has increased, particularly in the past month; though he has attended to his business, he has felt mean enough to want to go to bed directly after his evening meal. He now weighs 145 pounds, having lost 10 pounds in six months. Four months ago his friends said he looked pale, and he is sure that he is paler now than a year ago, but he doubts if he is any paler than four months ago. There have been no other symptoms.

Physical Examination.—The patient looks a trifle pale and appears tired. The complete examination is entirely negative except for the abdomen.

The abdomen is not tender anywhere. The lower border of the spleen is easily felt with the patient breathing normally 2 cm. above the level of the umbilicus; it extends 6 cm. below the costal margin in the left nipple line, and is 3 cm. to the left of the midline at a point 4 cm. above the umbilicus. It is hard, though it does not feel so hard as the spleen of the last case I showed you. The liver edge can be easily felt descending 1 cm. below the costal margin on inspiration. No other organs

or masses are felt. There is slight tenderness over the left lumbar muscles.

The left-sided pain and backache that the patient complains of are probably caused mechanically by the weight of the spleen.

All laboratory examinations are negative except for that of the blood.

The *blood* has been examined several times in the past two weeks, and has always shown essentially the same findings. The *red count* is 4,800,000 and the *hemoglobin* 80 per cent. The *white count* fluctuates between 13,000 and 18,000. In the *routine report* the red cells were noted as simply showing slight variation in size, and the platelets as about normal in numbers. The differential count was given as follows:

Polynuclear neutrophils	60 per cent.
Small lymphocytes	17 "
Large lymphocytes	4 "
Large mononuclears	19 "

From this report one learns nothing that definitely suggests what the condition really is. It was thought by some at this time that the case was one of early Banti's disease. This, however, may be essentially ruled out by the elevated white count alone. The elevated white count in the presence of an enlarged spleen, with a report of *19 per cent. mononuclear* cells, should make one suspicious that the reported large mononuclear cells may have been mistaken for some abnormal mononuclear cells. Such is the case in this instance. When this patient's blood was *critically examined* it was found that the differential count was essentially as given (0.5 per cent. eosinophils and 0.5 per cent. mast cells did occur), but that the 19 per cent. large mononuclear cells were nearly all abnormal mononuclear cells. About 8 per cent. were clear-cut definite *myelocytes*, though their granules were often finer than the usual text-book myelocytes; 3 per cent. were probably normal large mononuclear cells. The other 8 per cent. were cells that looked at casually appeared like certain normal large mononuclear cells. I am unable definitely to name these cells, but will say that they were large mononuclear

cells abnormal in the peripheral blood, and that they gave the oxydase reaction, showing they were of myelogenous origin. They varied in size from that of a polynuclear to over twice this size. Practically all had dark large vesicular nuclei, at times double. Their protoplasm often curled and contained fine granules, and often quite coarse ones. Sometimes the granules were short and rod shaped. These cells were highly probably some form of myelocyte. Rarely a very large mononuclear cell occurred with intensely deep blue protoplasm. The lymphocytic cells not infrequently showed some immature forms. The polynuclears were sometimes abnormally large, and again abnormally small, not infrequently containing single-lobed nuclei.

From this white cell picture alone one should realize that he is probably dealing with *an instance of myelogenous leukemia* in a stage where the total white count is but slightly elevated. Further evidence of this is suggested by the *red-cell picture.* The red cells at a first glance are not at all striking, but when more critically examined one is impressed with the amount of abnormality they show, particularly when one considers the high level of the hemoglobin and red count. There actually occurs a definitely abnormal variation in size; occasionally a real microcyte is seen. No real macrocytes occur or deeply stained red cells. There is slight achromia. Variation in shape occurs, but is relatively slight. However, at times a narrow elongated cell is seen, and the smaller cells are more abnormal in shape than the larger. They sometimes are somewhat oyster-shaped, and at times bizarre-shaped cells occur. Rarely fine stipling and occasionally polychromatophilia is to be seen; and rarely a *true normoblast.* The reticulated red cells are 3.5 per cent.

This evidence of disturbed erythropoiesis in the presence of a high red count and hemoglobin is frequently seen in myelogenous leukemia, and such a picture is, I think, probably never seen in Banti's disease. This red-cell picture with a high red count may occur in other conditions, but relatively rarely in others than polycythemia vera. In this connection it is inter-

esting to note that it has been suggested that polycythemia vera is the red cell homologue of the white cell condition known as myelogenous leukemia. I have seen 2 cases which have shown early in the course of their disease polycythemia, and later died with the pathology of myelogenous leukemia. I have also seen polycythemia vera in a woman whose sister has had classical myelogenous leukemia.

Further observations on the blood of this case before you showed that the *platelets* were definitely increased above their normal numbers, and were not infrequently abnormally large. An unusual number of these small important elements formed from the giant cells—megakaryocytes—of the bone-marrow is the rule in myelogenous leukemia. In certain instances they may be temporarily decreased. Sometimes I have seen in myelogenous leukemia, associated with a greatly increased number of platelets, true megakaryocytes in the peripheral blood.

From what has been said about the blood of this case, taken together with the fact that the patient has an enlarged spleen, the diagnosis of *myelogenous leukemia* is established. It is, of course, of an unusual type because there is no great elevation of the white count. Cases of this sort may occur with white counts under 9000; and in such cases careful blood examination, in contrast to routine examination, may alone be the method of differentiating the condition from Banti's disease. Some cases of myelogenous leukemia may run the whole course of their disease with relatively low white counts, while others may spontaneously exhibit at one time a low count and at other times a high count.

The fact that the blood condition was not recognized in this case (III) by the house officers doing the routine laboratory work is not because of their lack of care or knowledge, but because the condition is difficult to recognize. It may be only because of the clinical picture that we shall be led to study the blood unusually critically, and thus establish the diagnosis in such instances. This form of mistake, dependent upon true difficulty in judging correctly what one sees under a microscope, is of a much more pardonable nature than the mistake illustrated

in the last case (II), where the presence of a large spleen was apparently ignored and peptic ulcer was diagnosed instead of Banti's disease.

This case (Case III) should be given treatment by exposures to radium or the Roentgen rays. The possibility of splenectomy in such cases has been referred to when we discussed the use of splenectomy in general.

CASE IV

This fourth case with a large spleen is perfectly simple to diagnose, and I shall have time simply to comment on some of the symptoms and the treatment.

The patient is a woman thirty-five years of age, whose history is entirely unimportant except for the following: Eight months ago she began to feel tired, lazy and sleepy constantly, and has continued to feel this way ever since; she feels more so now than some months ago. The patient has lost 20 pounds in weight during this time, and now weighs 160 pounds. In the past six months she has had a poor appetite, and has had some mild epigastric discomfort, and a slight full feeling in the left side of her abdomen. Though an intelligent patient, she has not noticed any mass in her abdomen. In the past two months cramps have occurred at times in the calves of her legs. She never had these before. About three weeks ago she began having, and has had every few days since, sharp momentary darting pains running down both legs, particularly the left one.

The *physical examination* is essentially negative except for a large spleen that reaches the umbilicus and fills the greater part of the left upper quadrant of the abdomen.

The *laboratory examinations* are all negative except for the *blood*, which shows as follows:

Hemoglobin, 75 per cent. Red count, 4,900,000.

The *red cells* show slight achromia and a definite variation in size, with an occasional microcyte, but no real macrocytes. Variation in shape is slight. Polychromatophilia occasionally occurs, and rarely stipling. Rarely a normoblast is found.

The *white count is 300,000*. The differential count is:

Polynuclear neutrophils	42	per cent.
Polynuclear eosinophils	3	"
Polynuclear basophils	1	"
Myelocytes: neutrophils	35	"
Myelocytes: eosinophils	3	"
Myelocytes: basophils	1	"
Large mononuclear cells	3	"
Lymphocytes	12	"

The platelets are definitely somewhat increased in numbers.

The blood-picture gives us at once the diagnosis, namely, *typical chronic myelogenous leukemia.*

The *symptoms* this patient presents are the typical ones given in cases of this disease. It is a feature of the disease for the patients to give for their first symptoms weakness, fatigue, sleepiness, or feeling "dopey," or for these symptoms to be among their first symptoms. Such symptoms, you will recall, were a feature of Case III, as they are of this case (Case IV). Loss of weight is also a common feature, and this, combined with symptoms of fatigue, etc., should always make one consider the possibility of this disease. Though most cases present these two features, all do not.

Some cases first consult a doctor because of the enlarged spleen *per se,* or because of discomfort dependent upon the enlarged organ. Probably many cases do not consult a doctor for months after the disease has started because the onset is very gradual and the disease may progress considerably without the patient having any symptoms.

Such darting pains in the legs as this patient describes are not unusual in myelogenous leukemia. We have observed that such pains occur not only in the legs but also in the arms. They have, however, occurred more commonly in the legs, and are usually described as more intense in the left than in the right one—perhaps because of the enlarged spleen. Cramps in the muscles, as also complained of by this patient, are not unusual in these cases.

This patient should be treated by radium or Roentgen-ray therapy. Splenectomy is distinctly contraindicated. She will be referred to the Collis P. Huntington Memorial Hospital of Harvard University for radium treatment, because there is established there a special clinic for the care and treatment of

these cases. With proper radium therapy we can practically assure this patient marked improvement in her symptoms. It is highly probable that her sense of fatigue, sleepiness, etc., will entirely disappear, and that she will gain weight and become symptomless and feel well. The spleen, in all probability, will become much smaller, and the white count will be brought down to somewhere near normal figures; the percentage of myelocytes will fall, and the detailed blood-picture as a whole will appear much more normal, including a lessening of her anemia.

However, though this treatment will produce this effect in the vast majority of cases, particularly if treated relatively early, it does not cure the disease. To keep these patients in the best possible health it is necessary to keep them under close observation, seeing them every few weeks, and giving suitable amounts of radium from time to time. As the disease progresses a relapse occurs, and finally therapy is without significant effect, and the patient dies of the disease. This method of treatment does, however, frequently bring about a temporary remission of the disease, so that patients who are often in a distressing and sometimes in an apparently serious condition can be returned to a useful and functionally efficient existence for a period of many months to some years. Some degree of remission is brought about in practically every case.

The rare acute cases do not benefit by this form of treatment or practically by any other form of therapy. Cases of lymphatic leukemia are not benefited by radium treatments to the degree that the chronic myelogenous cases are. Not infrequently the lymphatic cases receive no particular benefit.

The cases of myelogenous leukemia that have the greatest symptomatic improvement following radium are those in which not only is the white count reduced but also the spleen much reduced in size. Cases in which the spleen is relatively slightly reduced, though the white count may be considerably or even markedly reduced, have, as a rule, a less satisfactory remission.

Through the character of the cells we can usually judge how the patient is doing—whether the process is progressing slowly or rapidly, or whether a stage of terminal chronicity is approaching or occurring, from which there will be no remission.

Though the white count perhaps is the best single guide to further treatment, as time passes by the character of the blood-picture, as well as the presence or absence of symptoms, is certainly equally and perhaps more important. For instance, if the white count has been reduced to well below 50,000, and then rises to this level with the patient feeling perfectly well, and the blood-picture shows essentially no abortive types of cells, and there is a low percentage of myelocytes, and especially if the spleen is small, it is probably fully as wise not to give more radium at this time as to give it. It is, of course, desirable that the patient be observed frequently under such conditions. It is advisable not to give more radium under such circumstances, because it seems as if too many constant exposures may cause in themselves undesirable features, while it is well known that distinctly too much radium may result in a fatal aplasia of the marrow.

If, on the contrary, the white count is 50,000 and the white cells exhibit certain abortive features—in which instance the patient would also probably be having symptoms—more radium may be given. Should such circumstances occur and the patient's spleen be relatively small rather than relatively large, the benefit will probably be slight.

The cases finally progress to a stage with increasing anemia where the blood-picture is of a distinctly abortive type, yet with the white count frequently under 80,000, and the spleen often not especially large. Under such conditions radium often fails to do what it has previously done for the patient; so the individual gradually fails and dies. Thus it is important to give the optimum amount of radium, and to treat the patient and not the white count. One always has to be guided between the amount of good that can be done and the amount of possible harm that may be done.

There are numerous points that I have not mentioned to you that could be profitably discussed about Banti's disease—its differential diagnosis, splenectomy, and myelogenous leukemia—but I have attempted in this hour simply to demonstrate to you certain significant facts about these conditions as illustrated by these 4 cases.

CONTRIBUTION BY DR. JOHN B. HAWES, 2d

MASSACHUSETTS GENERAL HOSPITAL

TUBERCULOSIS AND INFLUENZA

Necessity of a Sane Conclusion in Reference to the Incidence of Pulmonary Tuberculosis as a Sequel to the Late Epidemic of Influenza

THERE is prevalent throughout this country, both in the minds of the medical profession as well as the laity, a strong feeling that there is to be a great increase in the incidence of pulmonary tuberculosis as a sequel to the late epidemic of influenza. Boards of health and tuberculosis societies and other organizations are sending out bulletins warning the public in regard to this, while in certain localities the medical profession seems to be somewhat stampeded by this idea. It is of very great practical importance to come to some sane, even if not very definite, conclusion on this subject. Unfortunately, it is impossible to obtain as yet any exact facts and figures, and it will be only after at least a year has passed that we can have any real knowledge on the subject. It will then be too late to undo a certain amount of harm that is now already being done.

The average physician seems to feel that because influenza has been primarily a disease of the respiratory organs, it must on this account have a disastrous effect on a hitherto existing pathologic process in the lungs, tuberculous or otherwise. I admit that this is a perfectly natural deduction, but I do not feel that it is necessarily in accordance with the facts. I was firmly convinced that this would be the case myself, and expected to have many of my patients with quiescent and inactive pulmonary tuberculosis report to me that their disease had broken out into activity once more. I have been agreeably surprised,

on the whole, in finding that this has not been the case. I have seen many patients who have had tuberculosis and who lately have had influenza, more or less severe, who complained of an irritating cough, loss of weight, lassitude, often some fever, and often signs of moisture in the lungs. These patients have been greatly alarmed and much discouraged over what they believed to be renewed activity of their old tuberculous lesion. While I have invariably treated them as if this were the case, I have been impressed and much pleased that in the majority of cases the signs and symptoms soon disappeared. I am not at all sure but I have seen about as many patients complaining of these postinfluenza symptoms—cough, loss of weight and strength, often a slight fever, moisture in the lungs—who have never had tuberculosis and who did not have tuberculosis at the time, as I have those who have had healed or quiescent tuberculous processes.

In the Massachusetts State Sanatoria, out of 1079 patients, there was an incidence of 11.2 per cent. of cases of influenza, and a mortality rate of 1.1 per cent. With one exception our superintendents feel that influenza, even accompanied by pneumonia, seems to have done remarkably little permanent harm to the tuberculous process that already existed. Physicians at Saranac Lake, with whom I have recently talked on this subject, seem to have the same opinion. Not only was the incidence of tuberculosis rather low, but the permanent harm it seemed to do was comparatively slight. One would naturally explain this by taking it for granted that the patient, having developed a certain amount of immunity to tuberculosis, had also acquired some immunity at least against this other respiratory disease—influenza.

It is interesting to compare the comparatively low incidence of influenza among the tuberculous patients at our state sanatoria with that among a similar group of patients in other institutions where there was comparatively little or no pulmonary tuberculosis. Among 4855 such patients, inmates of five state institutions, there was an influenza incidence rate of 30 per cent. and a death-rate of 2.1 per cent., while at one large general

hospital where there were 175 nurses at the height of the epidemic in October, 48 per cent. of these came down with influenza. These figures certainly show fairly conclusively that influenza was less frequent and did less harm, as far as death was concerned, among consumptives than it did among others not suffering from this disease.

Dr. Donald B. Armstrong, director of the Framingham Community Health and Tuberculosis Demonstration, in a recent article entitled "Does Good Health Pay?" calls attention, in a little different way, to this same fact. In his investigations at Framingham he has noted that influenza seemed to have been distinctly more severe, and the mortality seemed to have been greater among those robust and strong individuals who have always enjoyed good health, while among those who have been weak, physically below par, whether suffering from tuberculosis or not, the disease has been milder and the toxins less virulent.

The diagnosis of tuberculosis, difficult at best, is certainly made increasingly so at the present time. Patient after patient has come to me, accompanied by distracted parents, relatives, or friends, who has been told that he has tuberculosis. Upon investigation, I have found many of these patients were merely suffering from the after-effects of influenza, and that there was not sufficient evidence upon which to base a diagnosis of pulmonary tuberculosis. The treatment, except that I did not send them to a sanatorium for consumptives, has been the same as if they had tuberculosis. The "Diagnostic Standards in Pulmonary Tuberculosis," prepared for the benefit of the Framingham Demonstration, and now widely used throughout the country, have, I believe, been of distinct value in clarifying our ideas on the diagnosis of this disease. Unfortunately, at the present time, even these standards, conservative and sane as I believe them to be, must be modified somewhat to suit present conditions. Dr. Arthur K. Stone recently told me, for instance, that, in his opinion, the man who made a definite diagnosis of pulmonary tuberculosis in the absence of a positive sputum or a definite history of tuberculosis prior to this epidemic was taking

a somewhat rash step, and was liable to do harm rather than good. With this opinion I am in hearty accord.

In my work among lung cases at the Massachusetts General Hospital, as well as in my own private practice, I have had constantly to unmake diagnoses of tuberculosis, based on what I believe to have been insufficient evidence. I frequently write on the patient's record, after a definite diagnosis of pulmonary tuberculosis had been made by some other physician, a note to the effect that "I do not believe there is sufficient evidence to make a positive diagnosis in this case," that "I do not believe he should be sent to a sanatorium, but that he should be kept under observation until the diagnosis is made clear, one way or the other." I am glad to state that in many instances I have seen the pulmonary signs clear up, and the patient's general condition return to normal in the course of a few weeks of careful observation and proper hygiene. Of course, no one can say, in such cases as this, that tuberculosis was not the cause of the patient's signs and symptoms, but, on the other hand, no one can say so soon after such an epidemic as this that a given patient's signs and symptoms were due to tuberculosis, and not merely after-results of influenza.

Therefore, until the contrary is proved, I do not think that it is wise to spread the report broadcast that there is sure to be a great increase in tuberculosis. While we certainly should be on the lookout for such cases, and should make repeated sputum examinations and keep all doubtful cases under observation, it is not fair to the individual patient nor to the public to take it for granted that signs and symptoms which under ordinary circumstances we would attribute to tuberculosis, and rightly so, are at present necessarily due to this disease.

The following cases emphasize certain points in this discussion, and show the need of conservatism and extreme care before making a definite diagnosis of pulmonary tuberculosis under present conditions:

CASE I.—G. F. A strong, healthy woman of twenty-four came to see me in January, 1919, with the following history: Her mother died of tuberculous peritonitis, and in one brother

there was some question as to his having had pulmonary tuberculosis. In December, 1918, while serving as a pupil nurse in a large hospital, she was suddenly taken with a pain in the left side of her chest. This gradually increased until she was obliged to go to bed. Two weeks later 500 c.c. of clear fluid were aspirated. She ran a temperature of over 100° F. while in bed, and runs a slight afternoon fever at present. The fluid has gradually decreased until at the present time there is practically none. Her general condition has steadily improved. According to the last *x*-ray and clinical examination there is no involvement of the lung itself nor any signs of fluid.

Here is a frank pleurisy with effusion, coming on suddenly in a remarkably strong and healthy woman, with, however, some tuberculosis in her family. According to the diagnostic standards mentioned above, "one should consider a typical pleurisy with effusion as presumptive evidence of tuberculosis." The question at once arises in this instance, whether or not this patient should be definitely stamped as having pulmonary tuberculosis. Personally, I should say no to this question. The treatment has been identically the same as if I believed it to be tuberculosis, and, I am free to admit, in dealing with a patient with as high degree of intelligence as in the present instance, it is a matter of almost academic interest alone as to whether or not we use the word "tuberculosis." Under other circumstances, however, one can easily imagine that it might be a tremendous blow to the patient and to the family to state definitely that this was tuberculosis, even if only in the mild form that these pleurisies usually take. Every physician who has been thrown at all intimately with influenza patients during this present epidemic has seen cases of pleurisy with effusion develop and quiet down, where the evidence was strong that it was the so-called "influenza organisms" (usually a strain of pneumococcus) and not tuberculosis that caused the pleurisy. I have not reported this case as tuberculosis, and I have told the patient and her family frankly that I do not believe that it is tuberculosis.

CASE II.—M. B. This man, a strong and healthy appear-

ing Jew, aged thirty, came to see me in December, stating that two months previous he had had influenza for two weeks. Shortly after this he spat up some blood—not as much as a teaspoonful in all. His sputum was examined by a local board of health, where it was reported to be positive. When I first saw him, aside from the fact that he had raised this blood, which was hardly enough to dignify with the name of a hemorrhage, and was said to have a positive sputum, he felt perfectly well. He had a normal temperature and pulse, and his general condition was excellent in every way. His throat was red and streaked with mucus. In the lungs I could hear absolutely nothing except an occasional dry râle. I told him that with a positive sputum he must, of course, consider himself as having tuberculosis, and must take the greatest care not to infect others. As he appeared to me to be perfectly well I allowed him to continue at his work. Two months later I saw him again. He was greatly disturbed because on account of a slight pain in his chest he had recently gone to a large public dispensary and was told that he must go to a sanatorium at once. Again his temperature and pulse were normal, his general condition excellent in every way, and he had gained 10 pounds in weight. This time, over the lower half of the left chest I found numerous sticky râles, but no evidence of consolidation or changes in the voice or breath sounds. I wrote on his record that this was not clinical tuberculosis. A month and a half later I saw him again, and could find only an occasional dry râle, and at my last examination I could find nothing wrong with his lungs whatsoever. I have examined his sputum myself and found it negative.

With a positive sputum, this case must be considered one of tuberculosis, although my own sputum examination has failed to reveal any trace of this disease. In his lungs I have at no time found anything that would even suggest tuberculosis, while from a general point of view he has been perfectly well. I insisted that he change his occupation—a baker—to something else where he would be of no danger to others, which he did. I am unwilling to call this a case of tuberculosis, and have told him that I do not think that any of his symptoms justify

this term. He is to keep under my observation at regular intervals in the future, and I believe understands the situation clearly.

CASE III.—J. R. O'N. This patient, a young man of twenty-five, was accepted as a student pilot in the aëroplane service in December, 1917. He caught cold in April, 1918, and was sent to an army hospital for thirteen weeks, where a definite diagnosis of tuberculosis was made. While there, he had a fever up to 102° F., with, however, a low pulse. Sputum examinations were constantly negative. He gave a history of having been sent away for a couple of months six years ago on account of his health, although no definite diagnosis was made. My examination in October, 1918, showed a normal temperature and rather rapid pulse, weight up to normal, and a right apex with slight dulness and signs of slight old fibroid tuberculous process. The lower half of the left chest in the back was dull, with diminished voice and breath sounds, signs of small amount of fluid and a thickened pleura. I told him that he should report once a month, and allowed him to continue his work. This he has done regularly. In January he had a mild attack of influenza and stayed at home a few days. Since then he has improved steadily; all that I can now find in his lungs are a few dry râles at the right base, where the influenza was localized. He is working every day.

Here is a man with an old quiescent tuberculosis, sufficient to cause his discharge from the army. He undergoes a mild attack of influenza, apparently without its causing any increased activity in his tuberculous process. Nevertheless, it would be very easy for anyone seeing this patient for the first time shortly after his influenza in January to have said that this was a case of tuberculosis roused into activity by influenza. I really do not feel that this has happened, nor do I feel that the influenza has done his tuberculous process any harm whatsoever.

CASE IV.—R. McN. This patient, a young man of twenty-one, came to see me March 1, 1919. His mother was said to have died of tuberculosis. He himself has always been strong and robust. In Christmas, 1918, he was taken with the influenza

and was in bed nine days. He went to work as a chauffeur shortly afterward, but about ten days ago had a hemorrhage of about an ounce of clear blood, followed in the course of the next twenty-four hours by the raising of a large amount of foul pus. At present he feels perfectly well and has no symptoms. Physical examination shows little, except slight dulness at the bases. *x*-Ray examination shows mottling over both upper lobes. Later examinations have shown absolutely no signs of activity in the lungs, while the patient himself feels perfectly well and is working regularly.

In this case there was apparently a slight tuberculous infection at the apices, as shown by the *x*-ray. The patient had never known of this, however, nor has it ever caused him any symptoms. After his influenza there was apparently a patch of what must have been an unresolved pneumonia, which broke down and was accompanied by the raising of blood, followed by pus. According to the diagnostic standards, "a hemorrhage of at least a teaspoonful of bright blood is presumptive evidence of pulmonary tuberculosis." Under ordinary circumstances, one would be justified in saying that this was probably pulmonary tuberculosis. Under present conditions, however, I do not feel that this is the case, nor do I feel that the influenza has had any effect on his quiescent tuberculosis.

CASE V.—F. S. This patient, a woman of thirty-one, had always been delicate, especially during the past five years. In January, 1919, she had a mild attack of influenza, and has had a cough ever since. Sputum has recently been found positive by a local board of health. Physical examination shows a woman in a marked state of what I believe to be postinfluenzal depression, with a subnormal temperature, low blood-pressure, rather rapid pulse, and some anemia. I could find absolutely nothing wrong in the lungs, despite the positive sputum. I have advised her to put herself at as nearly absolute rest as possible, and to let me see her in a month's time.

Here again we have a case with positive sputum on one examination. The symptoms, however, are far more those of the physical depression so characteristic of the recent epidemic

of influenza, rather than those of any active tuberculosis. This is confirmed by the fact that the signs in the lungs were conspicuous by their absence. Are we going to say that this woman has tuberculosis as a result of influenza, or are we going to take the more conservative course, and treat her as if she had tuberculosis, keep her under observation, and yet not add to her depression by using the term "consumption." To some this latter may seem a form of camouflaging the truth; to me it seems the only humane course to pursue.

CASE VI.—J. G. A physician in charge of the tuberculosis dispensary in another Massachusetts city made application for this man's admission to one of our state sanatoria. After looking over the application blank, I was in some doubt as to the diagnosis, and wrote to the doctor as follows: "I note that the signs are entirely at the bases of the lungs; likewise that there is a negative sputum, a normal temperature, and a fairly normal pulse. You state that this condition is a sequel of influenza. I would like to know on what you base your diagnosis of tuberculosis in this instance? There are so many cases where the signs are located at the bases of the lungs following this influenza which are called tuberculosis, but which really are simply the sequel to influenza and due to the same organisms which caused the influenza, that I am trying to weed out all doubtful ones." I received his reply to the effect that he considered this patient to have a general influenza bronchitis, but the fact that it persisted and that the man was not doing well made him fear that he might be doing this patient an injustice by not sending him to a sanatorium. I wrote at once, stating that I would gladly give this man a trial at the Rutland Sanatorium, hoping thereby not only to improve his general condition, but to clear up the diagnosis.

Were I not to follow up this particular case after his admission to the sanatorium to find out whether or not this was tuberculosis, this man would go down automatically on our list as a case of consumption following influenza. While in this instance it certainly will do no harm to send him to a sanatorium for consumptives, and will probably do him much good, I feel

that this course should be pursued only in exceptional instances.

Cases similar to these might be endlessly multiplied. I do not wish to give the impression that I believe influenza is not an extremely dangerous respiratory tract disease, and that it does not do a certain amount of harm in practically every case. On the other hand, I wish to go on record as being definitely of the opinion that not only do patients who have or who have had pulmonary tuberculosis possess a certain degree of immunity or resistance against influenza, but that in my experience, at least, influenza or influenza-pneumonia does not seem to have done the great harm to the already diseased lung that most of us, myself included, thought would be the case.

As I have stated before, I have seen and am seeing many patients whose old tuberculous processes have *apparently* been lighted up by an attack of influenza. I am constantly receiving applications from physicians all over Massachusetts for the admission of patients to our state sanatoria whose disease, according to the statement on the application blanks, has been lighted up and rendered active by influenza. My experience so far at least, and I admit that it is too soon to speak definitely on this subject, has been that in the case of many of these patients the extensive signs in the lungs following the influenza have rapidly cleared up, leaving the old tuberculous focus comparatively little worse than it was before.

A physician in New Hampshire recently told me that he was making routine examinations of all his influenza patients one or two months after the acute symptoms of the influenza had subsided. I believe that this is an excellent course to pursue. I believe that all doubtful cases should be sent to a sanatorium or other similar institution for study and observation. I do not believe, however, that we should take it for granted, as is being done in many instances, that signs in the lungs and constitutional signs and symptoms persisting long after an attack of influenza are necessarily due to tuberculosis.

CLINIC OF DR. FREDERICK T. LORD

MASSACHUSETTS GENERAL HOSPITAL

A PULMONARY DESTRUCTIVE LESION

A Case Teaching Exercise,[1] with Remarks on History-taking, Physical and Certain Special Examinations in Diseases of the Lungs, and the Diagnosis and Treatment of Pulmonary Abscess.

THE following case history represents an actual and urgent problem and may well serve the double purpose of material for an exercise in case teaching and the placing on record of an instructive pulmonary lesion. In the criticism of this or any record it should be the purpose in reviewing it to note what of consequence may be lacking and how the work might have been better done.

You are asked in your report[2] on the case to state (1) what more you would like to know, (2) the diagnosis, and (3) the treatment you would recommend. You need have no hesitation in expressing yourself freely in criticism of the records. Regarding the diagnosis you make, to speak frankly but without any ungracious intent, I may say that while I am pleased when your diagnosis is right, I take a certain satisfaction in your mistakes, from which I can judge your deficiencies. It is a rare opportunity to make a mistake to your own advantage and not to the detriment of the patient.

The case history for consideration is as follows:

[1] I have used the case history in my Course in Physical Diagnosis and Medical Practice for Graduates at the Massachusetts General Hospital, but the present discussion of the case was put together for the purposes of this article.

[2] The case histories are distributed before the exercise, and each member of the course is asked to hand in a written report.

History.—Miss Y., forty-three, born and living in Massachusetts, consulted me October 6, 1914. The history is reported as if taken today.

Concerning her family history, her mother died of tuberculosis when she was four years old, and she was exposed. Her father died of "creeping paralysis." A half-brother is well.

Of past illnesses, it may be noted that she had a gland in the left side of the neck thirty-three years ago. It did not rupture and was not operated. Over a period of six to seven months twenty-seven years ago she had a cough. She cannot remember whether during this interval she had fever, night-sweats, or loss of weight. She remembers that there was ulceration of both corneæ twenty-six years ago, but she cannot recall whether or not the term "phlyctenular" or "scrofulous conjunctivitis" was used in connection with this disturbance, and she does not know the cause of the ulceration. There is no history of hemoptysis or pleurisy. Following scarlet fever she had abscesses in the left ear. She has had measles. There was a nervous breakdown nineteen years ago lasting one year. Since the age of fifteen she has had hay-fever of the fall type. There is no history of chorea or rheumatism. While in Rome, three years ago, she had a neuralgic pain in the region of the left ear, lasting three days.

There is nothing noteworthy regarding her habits. She is not accustomed to take alcohol. The bowels move regularly each day. The catamenia is regular and lasts three days. The flow is not excessive and there is no pain. The last period was three weeks ago.

Regarding the present illness, she states that nine months ago, without preceding respiratory disturbance, she nearly fainted, and had chill, fever, and weakness. The fever lasted two to three days, 101° F. being the highest temperature. There was no cough, rhinitis, or sore throat at this time. At the end of three days she had severe pain in the left side of the head in the region of the left ear. The pain lasted twelve to fifteen hours out of the twenty-four, and continued for nearly three weeks. A week after the onset of the present illness she had the following:

After having been abed for about an hour and asleep or

dozing, there was a sudden and violent paroxysm of cough without expectoration or noteworthy dyspnea. The paroxysmal cough lasted for one to one and a half hours and during it there was a sensation of bad odor and bad taste in the mouth. She vomited without nausea during the attack. There has since been a recurrence of the paroxysmal cough without expectoration six times each day and slight cough in the intervals between the attacks.

Following this she went away, feeling badly and continuing to cough. During the next seven weeks her temperature was taken at irregular intervals and was found to be elevated for varying periods, once for as long as ten days, the variations in the temperature being from 99° to 100.6° F.

Seven months ago she awoke at 5 A. M. with intense pain in the left axillary region, much aggravated by cough and long breath. The pain lasted one week, and during this time the temperature was elevated, but always below 100° F.

A week after the onset of the pain there was a gush of about an ounce of green, foul-smelling pus, which came up with the cough, the cough having previously been without expectoration. Cough and expectoration have persisted since and evening elevation of temperatute from 99.2° to 99.4° F. continued until about six months ago, when she went to Georgia, where she continued to cough and expectorate, but was without fever.

About five months ago she had a second attack of pain, this time on the right side, lasting about two weeks.

Between three and four months ago for a period of about a month she lost her voice, and for part of the time could not speak above a whisper.

About three months ago the cough was less troublesome and she felt better, but after a walk she had fever and subsequent afternoon elevation of temperature off and on to two months ago, when she had a third attack of pain, this time on the left side, in the region of the scapular line and posterior axilla. The pain lasted for five days and was accompanied by fever. There has since, with the exception of a few days, been an evening elevation of temperature from 99.4° to 100° F., while the morning

record has usually been normal. The sputum averages about 3 ounces in twenty-four hours and is purulent and green in color. She coughs at frequent intervals during the day and raises something every time she coughs. Every few days the amount of sputum is greater, and on these days it usually has a bad odor and taste, while on the intervening days it is likely to be little or not at all offensive. The amount has never exceeded 1 ounce at any one time. About seven months ago the sputum contained a small amount of blood surrounding the purulent masses, and on two occasions since it has been bloody. About three weeks ago the sputum contained about 1 dram of blood, and a few days ago it was stained a brownish color.

The cough is paroxysmal, the attacks recurring at frequent intervals during the day. It is uninfluenced by position. She has been short of breath for seven months. She had night-sweats for about two weeks after each attack of pain. Her average weight is 130 pounds. The weight is now 134 pounds. There is no nausea or vomiting. The appetite is poor.

Physical examination shows a well-developed and nourished woman. The skin and mucous membranes are of good color. The pupils are equal and react to light. Their outline is slightly irregular. There are corneal scars in both eyes. Examination of the right fundus with the ophthalmoscope is negative. The left is not seen on account of the corneal scars. There is no glandular enlargement. The mouth shows many filled teeth, one carious stump in the left upper jaw, and some pyorrhea. There is a slightly musty odor to the breath. There is no tenderness over the sinuses. Examination of the ears with the otoscope is negative. The nasal cavities, the throat, and the nasopharynx show nothing abnormal. Inspection of the larynx with the laryngoscope is negative. As far as can be seen with this instrument, the trachea shows nothing abnormal.

The cardiac impulse is palpable in the fifth space, nipple line, with corresponding dulness 12 cm. from the midsternum. The right and upper borders are normal by percussion. The heart sounds are regular, of good quality, and there are no murmurs. The pulmonic second sound is greater than the aortic second sound, but not abnormally accentuated.

Inspection of the chest shows no asymmetry. Expansion of the left lung is slightly restricted. On palpation, the intercostal spaces are the same both sides. There is no abnormal supracardiac or spinal dulness. The apices of the lungs show nothing abnormal. Pulmonary excursion determined by percussion during full expiration and inspiration is equal and of good amplitude on both sides. In the left back in the paravertebral region, between the seventh space and the tenth rib and over an area extending laterally to the posterior axillary line, are the following signs: There is slight dulness apparently of the same degree throughout the dull area. There is also slightly increased and bronchial breathing, increase of voice (without egophony), increased whisper, and much diminished but not absent tactile fremitus. No râles are heard. There is no cracked-pot sound, amphoric breathing, tympany on percussion, or change in the percussion note on changing the position of the patient.

Examination of the abdomen shows nothing abnormal. The liver, spleen, and kidneys are not made out to be enlarged. The spine is negative. The knee-jerks are present and normal. There is no edema. Blood-pressure is 120 systolic and 80 diastolic.

The blood shows a white count of 10,600, of which 85 per cent. are polynuclear cells and 15 per cent. lymphocytes. Wassermann test on the blood is negative. The urine is normal, acid, 1028. It contains the slightest possible trace of albumin, but no sugar. The sediment shows a few squamous cells, an occasional leukocyte, and no casts.

The sputum amounts to about 3 ounces in twenty-four hours, is green and purulent, with a slightly musty odor. Three specimens are negative for tubercle bacilli. No actinomyces, blastomyces, or streptothrices are seen. There are numerous small organisms morphologically like influenza bacilli, many cocci, and a few bacilli. In one of three specimens there is a small curled, elastic tissue fiber, without alveolar arrangement. No tumor fragments or tumor cells are found. Five previous specimens of sputum are said to have been negative for tubercle bacilli.

x-Ray examination shows some peribronchial thickening and there are numerous small apparently calcified areas, and a small calcified spot at the right apex. In the left lower chest there is a well-circumscribed area beginning above at the eighth and below at the tenth rib, and extending outward to the axillary border, of oval shape, and measuring 4 inches in greatest diam-

Fig. 241.—

eter and 2 inches in width. There is no evidence of a foreign body.

Criticism of the Record.—*The History.*—The most important guiding principle in history-taking is always to try to make a preliminary diagnosis from the history alone. The first matter for consideration is whether the history is as complete as it should be. We are dealing here with a problem in pulmonary diagnosis, and in all such cases the possibility of tuberculosis should be constantly in mind. An inquiry has evidently

been made in this case concerning those features which have a bearing on this question. Then you will note that in the description of the present illness the evolution of the symptoms is given. In pulmonary problems the number of possible symptoms are few, and much importance may be attached to the order of their appearance. Many mistakes in the diagnosis of disease of the lungs as well as of other organs may be ascribed to insufficient attention to the march of events. The relation in point of time of hemoptysis to other symptoms is, for example, an important matter, and in this case it should be noted that there was a small amount of blood surrounding masses of sputum after the cough had lasted for about seven weeks, and frank hemoptysis after about six weeks of cough and expectoration. It is in general true that hemoptysis out of a clear sky, or when cough and scanty expectoration alone cloud it, is almost invariably due to pulmonary tuberculosis. Blood surrounding masses of mucus, however, can hardly be regarded as hemoptysis in the general acceptance of the term. In this case, therefore, the hemoptysis late in the illness is less suggestive of tuberculosis, but hemoptysis out of a cloudy sky is also often due to tuberculosis.

The evolution of symptoms is also of importance in many other pulmonary conditions, and among them pulmonary embolism and infarction, of which there are suggestive features in this case. In fact, the distinction between pulmonary infarction from a latent venous thrombosis and pneumonia can at times only be made by attention to the sequence of the symptoms. In pulmonary infarction pain in the side is the most constant and usually the initial symptom, and if there is a succession of emboli the pain may be first on one and then on the other side, due to embolism of one and then of the other lung. Pleural pain in the first instance on the left and next on the right side is somewhat suggestive of embolism and infarction in this case. When infarction involves an area of considerable size cough and bloody expectoration occur within a few hours to two or three days after the pain, and the temperature slowly rises, while in pneumonia the pain, cough, bloody expectora-

tion, and rapid rise of temperature occur at once or in rapid sequence. Small emboli may give rise only to pleural pain without subsequent cough and bloody expectoration, and in this case if embolism occurred the clot was probably too small to cause typical symptoms, but the record should have been amplified with respect to the events following each attack of pleural pain.

Physical Examination.—Are there omissions in the physical examination? Nothing is said of bronchoscopy. Bronchoscopy is to be considered from the point of view of its danger and the advantage to be derived from its use. Many lives which might be saved are lost each year in consequence of the aspiration of foreign bodies because of the slowness with which medical men have informed themselves of the progress made in the use of the bronchoscope, and it is essential that wide stretches of the country now unprotected be supplied with physicians skilled in its use. But its use is more particularly urgent in the period immediately following the inhalation of a foreign body to prevent the immediate and remote consequences. A bronchial tumor or an aspirated foreign body is to be considered in this case, and bronchoscopy might establish the diagnosis. A tumor could hardly be successfully removed by this means. The removal of a foreign body lodged so long in the bronchi would not be likely to relieve the secondary and chronic inflammatory changes, but might alleviate the symptoms. The bronchoscope is of itself not without danger, and the symptoms do not seem sufficiently suggestive or the prospect of relief hopeful enough to justify its use here.

The use of tuberculin is not mentioned, but the presence of fever may be regarded as a contraindication, since an elevation of temperature is the principal feature of the test.

Inoculation of a guinea-pig with the sputum would be desirable, but we should have to wait six weeks at least for the development of tuberculosis in the animal, and it would seem best to come to some conclusion regarding the problem before this time. This also was not done.

Irregularity in the outline of the pupils when accompanied by inequality and failure to respond to light is suggestive of

syphilis, but only irregularity is present here, and is probably due to a disturbance in the iris at the time the corneal opacities were formed. The pulmonary condition does not suggest syphilis, and it seems quite unnecessary to pursue the investigation further than the negative Wassermann test, which should be done as a routine. If syphilis could be entertained as a possibility then a numerical and differential count of the cells in and tests for protein and Wassermann on the spinal fluid should be done, but the withdrawal of spinal fluid is not a wholly indifferent matter to the patient and there should be some definite indication for its performance.

Regarding examination of the chest, I can perhaps help you by telling you how you can yourself get the most in the way of instruction out of your investigations.

Skill in percussion is usually the last and most difficult acquisition of the student. You should always do your percussion of the chest first. Light seems preferable to heavy percussion. Percussion should be rapidly done so that slight differences are more readily appreciated by an immediate contrast of abnormal with normal resonance. The mere finding of a dull area is not enough. Its limits and the character of the dulness throughout all parts of the region should then be determined, and it is of interest to know that the dulness was of the same degree throughout the dull area here. An error, commonly made in the percussion of dull areas at the lower limits of the lung, is to fail to note the degree of dulness at the extreme base. In the period during which we have controlled our chest findings with the *x*-ray there have been a number of cases in which the *x*-ray has shown an uninvolved portion of the lung between the upper limit of the diaphragm, and the dull area on percussion and reinvestigation has shown that greater care in the determination of the limits of the area by percussion would have disclosed this. Once you have learned the rudiments of physical examination, no one can teach you percussion with an advantage equal to your own in the immediate check on percussion by the use of the stethoscope. In the case under discussion the changes found with the stethoscope immediately confirm the

previous and independent observation of dulness. You should thus let one method serve as a check against the other to make the most of the opportunity.

There is no other recent addition to our methods in pulmonary diagnosis of equal educational value to the *x*-ray. Just as percussion may be checked by the stethoscope, so the final results of physical examination may be confirmed or not by *x*-ray examination, but to realize fully the educational value of the *x*-ray all physical signs must first be recorded. There are, however, limitations to the *x*-ray, and it cannot be expected to demonstrate certain shadows which are below the level of the dome of the diaphragm or behind the shadow of the heart. The *x*-ray findings in pulmonary and other conditions should be regarded only as part of the evidence, and a final conclusion reached only after full consideration of all the data.

A STUDENT: How do you interpret the physical findings in this case?

DR. LORD: The correlation of physical signs with pathologic lung conditions shows that there are two well-defined groups of signs corresponding to two groups of lesions. Dulness, bronchial breathing, increase of voice, whisper, and tactile fremitus are found when there is increased density of the pulmonary tissue and patent bronchi within the consolidated region, as in ordinary lobar pneumonia, bronchopneumonia, pulmonary infarction or retraction, and compression of the lung. Dulness, diminished or absent breathing, voice, whisper, and tactile fremitus are common to all lesions where there is increased density and lack of communication of the involved region with the bronchi, as with an accumulation of fluid or a tumor in the lung or pleura, cysts, atelectatic areas from bronchostenosis, and massive pneumonia. The signs here do not fall into either group and suggest a combination of a member of one with one of the other group. The dulness, bronchial breathing, increased voice, and whisper suggest a consolidation of the lung with patent bronchi due to pneumonic infiltration or infarction, and the diminished tactile fremitus, in addition, suggests fluid or solid in lung or pleura without free communication with the

bronchi. The absence of râles suggests that pneumonic infiltration if present is relatively inactive and probably of the indurative variety. From the signs alone no further conclusion as to the nature of the process seems justifiable.

Such evidence against cavity as the absence of cracked-pot sound, amphoric breathing, and tympany on percussion are of no value in excluding it. A small cavity or small multiple cavities, if present, would not be likely to show any of these signs. Much depends on the position of the cavity, whether deep or superficial, and the freedom of communication with the bronchi. The smallest cavity detected as such in our series measured at autopsy 3 by 2 cm. and was just under the pleura in the posterior aspect of the left lower lobe.

Is there anything further you would like to know about the sputum?

STUDENT: I should like to know if it separated into three layers on standing.

DR. LORD: Why?

STUDENT: Because it would then have a bearing on the diagnosis of bronchiectasis.

DR. LORD: The sputum from patients with bronchiectasis will often separate into three layers on standing. The separation into layers depends on the admixture of part of the sputum with air and mucus, so that there is an air-holding frothy layer containing mucopurulent masses at the top, a thin cloudy fluid in the middle, and a sediment of pus at the bottom. But this appearance is not peculiar to the sputum with bronchiectasis, and is seen with pulmonary abscess and the evacuation of empyema through the bronchi. I do not think stratification of the sputum is of diagnostic value.

An omission may be noted in the *x*-ray examination. If *x*-ray of the left mastoid showed increased density it would help to explain the history of pain in the ear three years ago, and again at the onset of the present illness, but would not solve the question whether lateral sinus thrombosis and pulmonary embolism were concerned in the lung disturbance. No *x*-ray of the skull was taken in this case.

There is something lacking in the description of the *x*-ray findings. Can anyone suggest what is wrong?

STUDENT: Was the *x*-ray taken with the patient in the upright or the prone position?

DR. LORD: That is an important matter. Why?

STUDENT: Is it not true that *x*-ray plates in such cases should if possible be taken with the patient in the upright position in order not to miss the appearance of a cavity with fluid level?

DR. LORD: Yes, and that is one defect in the report. Is there any other?

STUDENT: I should like to know the character of the area, whether there is in this region mottled uneven density or an unevenly dense shadow and the degree of density.

DR. LORD: This is also an important inquiry. In answer to the first question, the *x*-ray plate was taken with the patient prone and there would then be no evidence of fluid level. The importance of position in the *x*-ray diagnosis of lesions in which there may be losses of pulmonary substance with fluid free to show a fluid level was not fully appreciated at the time this patient was under investigation. If air and fluid are present in a pulmonary cavity, the central bright area due to the air may be entirely obliterated in *x*-ray plates taken in the prone position, on account of the diminished radiance of the fluid through which all the rays which enter the cavity must then pass.

In answer to the second question, there was an even density of marked degree over the circumscribed area as shown in the photograph. It is important to note this because by making the distinction between mottled, uneven, and evenly dense shadows evidence may be obtained concerning the nature of the process. Mottling suggests a varying degree of infiltration such as may occur in bronchopneumonia. An even density of considerable degree suggests a circumscribed collection of fluid or solid, but it is usually unsafe to make a diagnosis of the nature of the process from the *x*-ray alone.

Still another omission in the description of the *x*-ray findings is the failure to note the amplitude of the diaphragmatic excursion during inspiration as seen with the fluoroscope. It would

be of interest to know this to confirm the finding on physical examination of a normal respiratory motion of the pulmonary margins, and to furnish additional evidence regarding the condition of the left pleura—whether adherent or free.

Etiology.—We have to consider as a cause pulmonary tuberculosis, bronchial or pulmonary tumor, aspirated foreign body, and pulmonary infarction.

The possibility of tuberculosis is suggested by the disease in the family and the opportunity for contagion. The glandular swelling in the neck many years ago and the corneal ulceration, possibly of a phlyctenular type, may be of significance in this connection. There is nothing specially suggestive of tuberculosis in the history of the present illness other than that any pulmonary disturbance calls for its exclusion. The lack of apical involvement on physical and x-ray examination is against it, but there are occasional instances of tuberculosis involving the hilus region, and no certain conclusions can be drawn from the site alone. The failure to find tubercle bacilli in specimens of sputum containing elastic tissue is of great importance and goes far toward excluding tuberculosis. We should expect to find tubercle bacilli with pulmonary destruction of such a degree as to give rise to elastic tissue if tuberculosis were the cause.

Bronchial or pulmonary tumor would explain the bloody sputum. The hilus region is a favorite site for neoplasm. Partial occlusion of the bronchus leading to the involved region, consequent stagnation of bronchial secretion behind the obstruction, secondary infection, with bronchiectasis, single or multiple pulmonary abscesses, and interstitial pneumonia would account for the entire picture and cannot be excluded. Tumor fragments and tumor cells were not found in the sputum, but are usually missed when tumor is present. The absence of metastases in accessible regions is not against tumor, but after a duration of nine months one might expect neighborhood involvement and evidence of this in the x-ray examination of the mediastinum, or in radial projections from the involved pulmonary region along the course of the larger bronchi. Bronchoscopy would help in the diagnosis. The duration is too long for a malignant growth,

as most cases are fatal within a year of the onset of symptoms, and after nine months this patient is still in good general condition.

An aspirated foreign body can neither be affirmed nor denied, but the aspiration does not usually take place insidiously in adults, though it may occur during sleep. The initial symptoms are paroxysmal cough, dyspnea, a sense of suffocation and cyanosis, followed after an interval of a few days by the secondary manifestations of pulmonary infection. In this case the symptoms of infection—chill and fever—came first and the cough later. The *x*-ray showed no foreign body, but certain foreign bodies are not opaque to the *x*-ray. Bronchoscopy would help in the diagnosis.

Pulmonary embolism must be considered. There is a history of abscesses in the left ear after scarlet fever. Three years ago there was pain in the left ear for three days. Early in the course of the present illness there was again pain in the region of the left ear as a prominent symptom. A latent thrombosis of the left lateral sinus from which septic material separated and found lodgment in the lung as an embolus would adequately account for the pulmonary symptoms. This explanation finds some support in the second attack of pleural pain which occurred five months ago. While the first attack of pleural pain seven months ago and the third attack two months ago may be attributed to extension of inflammation from the left lung to the corresponding pleura, and the right-sided pleural pain may also be due to pulmonary infection proceeding from the lung itself, yet invasion from without, as from a venous thrombosis, is a more likely explanation. Ophthalmoscopic inspection of the veins of the fundus is desirable where thrombosis of the lateral sinus is suspected, since thrombosis of the lateral may lead to interference with the flow of blood in the cavernous sinus and engorgement of the ophthalmic veins. Unfortunately, the corneal scars prevented the examination of the fundus of the left eye. *x*-Ray examination of the left mastoid is also desirable, but was not done in this case.

Diagnosis.—Bronchiectasis is a possibility. It is an unusual

condition and may be suspected in the presence of any chronic pulmonary infection, but is incapable of clinical proof. To raise bronchiectasis often to the position of an independent disease is to fail to appreciate the pathology of the condition. Aside from its occurrence in the rare cases of bronchial asthma without secondary infection it is never found at autopsy unassociated with pulmonary changes of much greater importance than the bronchiectasis itself. Chronic infection of the bronchi is not likely long to exist without extension to the neighboring pulmonary tissue, and a more accurate term is then a "chronic bronchopulmonary infection."

Although the cause cannot be determined, the data point to pulmonary abscess, the five cardinal signs of which may be enumerated as follows:

1. Foul sputum and foul breath.
2. Cough and explosive expectoration.
3. Elastic tissue with alveolar arrangement in the sputum.
4. Dulness on percussion over a circumscribed area.
5. The demonstration by *x*-ray examination of a cavity with fluid level.

The five cardinal signs are not wholly fulfilled. To be certain of a pulmonary origin of the elastic tissue an alveolar arrangement must be demonstrated, and this was not present in this case. Elastic tissue without an alveolar arrangement may come from the pleura or the bronchi as well as the lung. The *x*-ray description does not indicate a cavity with fluid level. It is essential that the plates be taken in the upright position to demonstrate fluid level and, as already explained, the *x*-ray was taken in the prone position in this case. Even in the upright position fluid level would not be shown unless there were both air and fluid in the cavity, and cavities unruptured or completely full of pus would show no fluid level. The necessary conditions seem sufficiently fulfilled, however, to make the diagnosis of abscess amply justifiable. It would seem indeed that less evidence than we have would do, and we often have to be satisfied with less than the five cardinal signs.

The diagnosis would not be complete without an attempt to

go further and define the pathology of the condition. The long duration of the suppurative process suggests that pulmonary induration is likely to have taken place about the site of the destructive process. Even though the area is circumscribed and evenly dense by *x*-ray examination, there may be multiple pockets within the site of involvement. The even density of the *x*-ray shadow, however, is more suggestive of a single abscess. It is important to know whether the pleura is free or adherent over the site of the abscess. Invasion of the pleura is suggested by the history of pleural pain, but the normal amplitude of the pulmonary excursion makes it improbable that obliteration of the pleural sac has taken place.

Prognosis.—What is the probable outcome of this case? The theoretic mortality in pulmonary abscess is 74 per cent. Of the cases which do not terminate fatally, 18 per cent. continue to cough and expectorate, and are constantly subject to the danger of local or general septic complications with their menace to life. In the remaining 7 per cent. complete spontaneous recovery may occur. The small group in which spontaneous recovery may occur, however, comprises only mild and uncomplicated cases of short duration. I have never seen a patient with a history of nine months' duration completely recover from abscess spontaneously. In this case, therefore, there is, so far as I can estimate, great danger to life, an almost certain prospect of continued invalidism, and practically no chance of complete recovery.

Treatment.—There is no satisfactory medical treatment. Quinke's position may be tried, but has not proved successful in my experience. There is but one promising method, and that a resort to operation. What are the dangers? It is a difficult and dangerous operation. It should be undertaken only by one skilled in its use, and even then the results are far from satisfactory in many cases. The operative mortality is high and may be placed at 6 deaths (9 per cent.) in 62 operated cases. Including cases in which the abscesses cannot be found, surgical failure may be estimated at 14 per cent. Surgical success is more likely in the acute than in the chronic cases. The delay

in seeking operative relief in this case has somewhat increased the chief danger of the operation—that of fatal postoperative hemorrhage due to the injury of granulation tissue in the wall of the abscess or of blood-vessels lining the wall or traversing the lumen of the cavity. Delay has also diminished the chance of complete cure because the firmer the pulmonary induration about the abscess, the less likely is the cavity to close after the establishment of drainage. Multiple pockets arising in the course of time by extension into neighboring tissue may make surgical drainage impossible. Bronchiectatic dilatation of bronchi communicating with the cavity may add to the surgical difficulty. Then there are the dangers of postoperative pneumothorax and empyema. The probable lack of pleural adhesions of sufficient density to withstand the operative manipulation increases the operative risk and makes it desirable that an operation, if performed, be done in two stages.

What are the chances with operation? Complete and permanent cure of abscess by operation may, in general, be estimated from our experience at 10 in 62 cases, or 16 per cent. Complete or partial success may be calculated at 38 per cent. In view of the absence of any obvious present complication and the sharply circumscribed and even density of the abscess by *x*-ray examination, and in spite of the long previous duration, the present situation seems as favorable as the general average. If not complete, then partial success may be hoped for in this case. What shall we advise this patient to do? There can be but one answer, and that is to take the risk of operation for the sake of complete or partial relief.

Operation.—Operation (Dr. C. L. Scudder) in first (October 17, 1914) and second (October 24, 1914) stages evacuated about 1 ounce of foul pus from a pulmonary cavity with indurated walls in the left lower lobe 2 or 3 cm. from the surface of the lung. A left pneumothorax developed after operation. Following this the cough and sputum became much less troublesome and the foul odor to the sputum disappeared, but a sinus with a slightly foul discharge persisted. The temperature became normal. A third operation in November, 1915, and a fourth

in May, 1916, were performed. At the last operation the pulmonary cavity was found to be about the size of the end of the middle finger. It communicated with two dilated bronchi, the size of a slate-and lead-pencil respectively. The cavity was cureted. The dilated bronchi were dissected free and tied off. The patient has since been well and without cough or expectoration.

Comment.—The diagnosis was correct in that the patient had a pulmonary abscess. The two dilated bronchi were not diagnosed and could not have been diagnosed. The etiology of the abscess is not known, but septic emboli from thrombosis of the left lateral sinus seem the most likely explanation. The operation was finally a complete success. The localization of the abscess was accomplished without difficulty in this case. Difficulty in sufficiently accurate localization of abscesses for successful surgical approach has hitherto been a troublesome matter, which an improved, portable, *x*-ray apparatus, enabling the surgeon to guide his search by means of the fluoroscopic screen, may obviate. The danger of postoperative pneumothorax, constantly present in operations on the lung through the unadherent pleura, is somewhat diminished by the use of one of the positive pressure methods of anesthesia, more commonly employed now than then.

CONTRIBUTION BY DR. H. LINENTHAL

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THE RELATION OF THE CLINICIAN TO INDUSTRIAL MEDICINE

IN a recent lecture at the Harvard Medical School Dr. Harry E. Mock outlined the growth of industrial medicine and pointed out the economic value to the community from the development of the industrial physician, who is becoming at present a highly specialized man. Up till recent times industrial medicine was confined to the work of the factory surgeon, who administered first aid in case of accidents. Very soon some physicians who entered this field of work recognized the great possibility of service that could be rendered to the employees, employers, and the community. The work of the industrial surgeon, in many industrial plants in this country, has very rapidly grown, and from the mere first-aid treatment the work has spread into many ramifications, such as the physical examination of applicants for employment, to determine the suitability of the particular individual for the particular job, thus rejecting the unfit before they start; the routine examination and treatment of those already employed; the supervision of the sanitary conditions of the plant, and many other lines, all calculated to promote the health of those employed, increasing the industrial efficiency, diminishing labor turn-over, which is a great drain on industry, and preventing the particular establishment from bearing the burden of those physically unfit.

To meet this ever greater demand for such health supervision in industry the position of industrial physician is rapidly developing into a specialty with a wide and most useful field of service. For in the present world crisis all movements tending

to the conservation of human life and safeguarding of human efficiency are of enormous importance.

But the study of the relation of industry to health, the recognition of the effects of industrial hazards upon the health of those exposed to them, is not a field for the industrial physician alone, in fact, it is not even primarily his field.

This field of study, the estimation of the part the patient's occupation had on the development of the disease for which he presents himself for treatment, is a field primarily for the physician who has to diagnose diseased conditions and treat patients in the hospital or in private practice.

Not only is the effect of occupation on health a matter of importance for the physicians in the diagnosis and treatment of the individual case, but inasmuch as occupational diseases are, in a large measure at least, preventable, the physician has certain duties to the community in the control of occupational diseases.

His duties and responsibilities in the control of occupational diseases are not in any way different from his duties and responsibilities in the control of communicable diseases. In this group of diseases also he should be the vanguard of the public health authorities, who, acting on his prompt diagnosis and report of cases, may take the proper measures to prevent the occurrence of other cases.

It is to the importance for the general medical man of recognizing the effects of occupation on health that I wish to direct attention. This field so important from the standpoint of the individual worker and the community remained long neglected by the medical profession as a whole. Interest on the part of medical men in this field has been rather sporadic. Most of the advances made till very recently have been made by sociologists, legislators, and economists, to whom the subject was ever of great importance. The medical profession as a whole has remained rather indifferent.

But very recently there has been a general awakening to the medical importance of the subject, and an intense interest is being manifested in various quarters in industrial diseases.

One of the factors which more than any other has helped to awaken this interest is the fact that the subject is receiving, as it were, academic sanction in many medical schools, notably at Harvard, where Dr. Edsall has been repeatedly emphasizing the importance of this field, and where a special department of Industrial Hygiene has been recently started and a journal devoted to industrial hygiene has been created.

Investigations into the effects of industrial hazards upon the health of the operatives exposed to them can, moreover, be made very fruitful, and should appeal to the research worker, for while we possess a considerable body of knowledge of the effects of industry on health, such knowledge is small compared with the problems which still await solution.

We have, it is true, some definite knowledge of the well-defined specific industrial diseases—the various industrial intoxications; the postural strains and localized fatigue effects manifested in the occupational neurosis.

We know, however, considerably less of the effects on the health of the operatives exposed to hazards which do not produce specific industrial diseases, but which nevertheless produce states of ill-health in which the industrial hazard clearly plays a prominent etiologic part. I refer to the hazards of non-poisonous dusts, of non-poisonous but irritating fumes, of extremes of temperatures, and of unsanitary conditions under which work is carried on, conditions which are not an integral part of the industry, but which are accidentally associated with it.

Our knowledge is still more meager when we consider the general effects on health from the factors more or less common to all modern industry—the stress; the speeding up caused by the machine-set pace; the monotony, mental and physical, caused by the great division of labor; and what seems to me is of vast importance, the general fatigue effects of industry. That these factors have no immediate effects that can be readily perceived and measured we know. They do not produce sudden, severe, incapacitating illness. They are not dramatic in their manifestations. But are they for that reason without effect? May not these factors to which the worker is exposed

throughout his working time gradually and insidiously undermine health and be responsible for the early development of the degenerative diseases of middle life—diseases which are coming more and more to the front as public health questions?

The limitation of our present knowledge of the effects of occupation on health is determined in part by the limitations of our physiologic and pathologic knowledge of certain problems; and in perhaps still a larger measure by our poor method of book-keeping. We have not kept accurate record of the relation of occupation to the train of symptoms which the worker complains of when he presents himself for treatment. For in our morbidity and mortality statistics we have not kept reliable records of the occupations. While death certificates provide for a statement of the occupation of the deceased, and hospital records contain statements of the occupations of the patients, the data are recorded in so unsatisfactory a manner as to give no indication of the actual work the deceased or the patient were engaged in. The general designation of the industry most often found on records, such, for instance, as rubber worker, shoe worker, textile worker, is not sufficient, for in the great variety of processes in any industry some have very definite health hazards, while others have no such hazards. To be of any value not only should the general industry in which the patient is employed be recorded but also the exact process the patient is engaged in.

This method of recording both industry and occupation has been adopted at the Massachusetts General Hospital in recent years. This detailed statement of the patient's occupation is in itself a great advance, and will in the course of time permit an analysis of the records according to the kind of work the patients were engaged in, and will permit deductions as to the relative frequency of certain diseases among any given class of workers. Were such a classification adopted by all hospitals and registrars throughout the country, it would in a very short time contribute an immense amount of knowledge as to the relation of industry to health.

A glimpse as to what interesting facts might be brought out

by an analysis of such statistics might be had by reading the summary made by Wynne and Guilfoy of the mortality in various occupations as shown by the returns to the New York Board of Health for one year. Small and insufficient as the data is, there are nevertheless many extremely interesting suggestions which point the way to further research.

Moreover, the recording accurately of the patient's occupation is not only important in hospitals, where, owing to the large numbers treated, statistics indicating the frequency of certain diseases among certain groups of operatives may be obtained, but is of great importance to the physician in handling his private practice, particularly when one practises in an industrial community.

A clear understanding of the patient's work will very often give a clue to the diagnosis of a case otherwise obscure, and frequently make an early diagnosis possible. The prompt and early diagnosis of occupational diseases is a matter of great importance both to the patient and to the community. To the former because prompt diagnosis will in most cases remove the worker from further exposure to the injurious agent, and thus minimize the possibility of very serious and even fatal injury. To the latter, that is, the public, the early diagnosis is important because occupational diseases are in a great measure preventable, and the prompt discovery of a case will, if proper measures be taken, prevent the occurrence of other cases. This responsibility of detecting the early cases and thereby preventing the occurrence of new cases rests entirely with the physician.

In 1914 in a paper on "The Early Diagnosis of Lead-poisoning" (Jour. Amer. Med. Assoc., June 6, 1914) I cited as illustrative examples a number of cases gathered from the Outpatient Records of the Massachusetts General Hospital which showed definite symptoms of lead-poisoning, and which, taking into consideration the lead hazard to which the patients were exposed in their work, should have made a diagnosis of lead-poisoning possible, and yet no such diagnosis was made because of the absence of a lead-line on the gums and of basophilic granules in the red blood-cells.

Since 1913 efforts at systematic study of industrial diseases have been made at the Massachusetts General Hospital. The work developed to such an extent that under the guidance of Dr. Edsall a special Industrial Clinic has been established, of which Dr. Wade Wright was in charge. When Dr. Wade Wright left for service the clinic was temporarily closed, but was recently reopened under the charge of the writer.

A trained assistant is placed every morning at the admitting desk of the Out-patient Department, and all cases employed in industries where there are any hazards are sent up to the Industrial Clinic for study. Special attention is thus given to the study and diagnosis of industrial diseases, which results in the discovery of cases which would have otherwise escaped attention in the general clinic.

In contrast to the cases referred to above, where diagnosis was not made owing to the failure to consider the hazards to which the patients were exposed, I wish to give you a few illustrative cases from the Industrial Clinic of how the diagnosis was frequently made because of the consideration of the hazard:

A man of forty-eight comes to the hospital complaining of attacks of epigastric pain, loss of appetite, and for the last ten days of diarrhea. There was no lead-line on the gums. Investigation disclosed the fact that as a shipbuilder he had been working for ten weeks in a lead-lined cold-storage room, and in his work he was exposed to lead dust and lead fumes. A diagnosis of lead-poisoning was made, which was confirmed later by finding lead in the urine and stools.

A plumber of forty-seven complains of attacks of abdominal pain radiating to the back. His appetite is poor; he has lost weight and he has pains in various joints. In spite of the absence of a lead-line, the symptoms and the lead hazard of his occupation pointed to a diagnosis of lead-poisoning. The diagnosis was later confirmed by finding lead in his stool.

A "spreader" in a rubber shop complains of attacks of abdominal pain which last a few minutes. He is very constipated.

A shipyard worker of thirty-five complains of pain in the lumbar region for three months.

A painter of thirty-one complains of loss of strength in his wrists and pains in his back and feet.

A painter of forty-eight complains of attacks of abdominal pain associated with constipation and dizziness.

An iron foundry worker of fifty-three has attacks of abdominal pain and constipation.

In all the above cases there were no lead-lines found, but inquiry into the occupations indicated the real lead hazard. The diagnosis of lead-poisoning was therefore made, and in every instance it was later confirmed by the finding of lead in the urine or stools, or both.

These cases illustrate the importance of considering the industrial hazard in the making of the diagnosis. Most of these cases would probably have escaped the diagnosis of lead-poisoning in a general clinic.

Unsatisfactory as is the situation in regard to lead-poisoning, it is still more unsatisfactory with regard to the less-defined industrial poisons. Absolute failure to correlate symptoms complained of by patients with the hazards of their occupation, even when such symptoms could not be explained by any of the physical findings, and would become perfectly clear if the industrial hazard were considered, was manifested to me several years ago when I looked up a number of patients exposed to naphtha fumes at their work.

Suppose a man presented himself to his physician complaining of attacks of dizziness, weakness, loss of energy and ambition, he tires easily, his appetite is poor, he has ringing in his ears, his speech is blurred at times, and he feels that he is losing his memory. Suppose the physician does not find anything on physical examination, but on inquiry into his patient's habits he learns that he is addicted to the naphtha habit—that to satisfy a perverse craving the patient inhales for several hours every day the fumes of naphtha. Would any physician fail under such circumstance to tell his patient that it was this perverse habit which brought about his state of ill health? Yet a man who actually presented himself with the above symptoms and who instead of inhaling naphtha fumes several hours

every day, inhaled it ten hours every day for six days in the week, not because of a perverse habit but out of necessity—he was cementer in a rubber coat factory, and while at work he was constantly bending over an open pot of naphtha cement—yet no diagnosis was made. The physician did not correlate the man's occupation and its health hazard with the symptoms of which the patient complained. All the symptoms cleared up on change of work.

A treer in a shoe factory suffers from dizziness, headaches, is sleepy and listless. Physical examination was entirely negative except for a mild secondary anemia. He inhales naphtha fumes, but the doctor who saw him did not realize the hazard, and made a diagnosis of constipation.

A driller in a watch factory complains of weakness, dyspnea, dizzy spells, loss of appetite, and insomnia. He has attacks of sneezing. A throat specialist, who saw him in consultation, gave as his opinion that the man suffered from "a peculiar vasomotor rhinitis," and the diagnosis made on the case was "constipation plus vasomotor rhinitis." Neither the throat specialist nor the medical man inquired as to what his work consisted of. If they had, they would have learned that to wash the plates he is working on, he squirts benzine from an oil can, and that many other men in the same room do the same work, and that the ventilation is inadequate to take care of the air so heavily charged with benzine fumes. Benzine-poisoning is a more intelligent diagnosis, one that explains the symptoms and the vasomotor disturbance, and one that would be likely to be of greater benefit to the patient.

A cementer in a rubber factory for six months complains of undue fatigue, headaches, nausea, and muscular twitchings. No diagnosis was made. At his work this man stands in front of an open bowl with naphtha cement.

A worker in a shoe-polish factory for six weeks, has not felt well for four weeks. Frequent attacks of vertigo, several attacks of vomiting; he suffers from frequent micturition. No diagnosis was made. This man was exposed to the fumes of turpentine at his work.

A man of twenty-three who worked as shoemaker in a rubber factory for two years, complains of headaches, attacks of dizziness, loss of appetite, bad taste in mouth, and constipation. These symptoms have been going on for a year and a half, but have become worse recently. The diagnosis that was made was that of constipation. This man uses naphtha cement constantly at his work. He works in a large room with 500 other workers in the same room, doing the same kind of work. The room is very warm. The windows cannot be opened for fear of drying the cement. It is quite evident that this constant exposure to naphtha fumes explains the symptoms perfectly well.

It is not necessary to multiply cases. The few I have shown sufficiently illustrate the evident fact that the failure to make a diagnosis in the above cases was due to the failure to take into account the occupation of the patients.

But the relation of industry to health is far broader in its scope than the specific diseases produced by industrial poisons. For, after all, the number of workers in industry who are exposed to industrial poisons is very small besides the vast army of workers who are exposed to dust and fumes and other conditions inimical to health.

Hoffman estimated that we have in this country at least 5,600,000 workers employed under conditions detrimental to health on account of atmospheric pollutions with dust and fumes, hazards which predispose or accelerate the development of tuberculosis and other diseases of the respiratory organs.

In a disease as wide-spread as tuberculosis it is, of course, difficult in any given case to state definitely that it is of industrial origin. The fact, however, that workers in certain industries give a higher death-rate from tuberculosis than other classes of workers living under similar conditions demonstrates very definitely that certain cases of tuberculosis are the results of industrial hazards. To take an illustration from Massachusetts' conditions. In the volume entitled "Tuberculosis in Massachusetts" Dr. Gordon, of Quincy, made a study of the occurrence of tuberculosis among the granite workers in that city. In a period covering ten years he found that the percentage of the

total number of deaths due to tuberculosis among granite workers was 46, while that of the rest of the community was only 16, which definitely demonstrates the very high death-rate from tuberculosis among that group of workers.

Dr. Gordon, however, made the extremely interesting observation that the mortality from tuberculosis among granite workers occurs later in life than is usual for the mortality from that disease among the rest of the community.

This condition is similar to the age distribution of deaths among miners, and quarrymen in the registration area of the United States, in that the highest mortality from pulmonary tuberculosis occurs among these workers between the ages of thirty-five to sixty-four, instead of between fifteen and thirty-four, the usual period of excessive mortality from tuberculosis.

If we consider, however, the mode in which the granite workers are affected, the reason for this high mortality from pulmonary tuberculosis later in life becomes clear. Stone-cutting is rather hard work and does not appeal to the physically weak. Those who start at the work are, therefore, apt to be in very good physical condition, and hence would have a natural immuntiy to tuberculosis. Long exposure to the irritating stone dust finally begins to produce changes in the lungs, the elastic lung tissue is replaced by scar fibrous tissue. This takes place gradually and insidiously, and may go on for a long time without giving rise to any symptoms. The fibrosis of the lung diminishes the resistance of the tissue and forms a favorable soil for the implantation of the tubercle bacillus. It is only then that infection takes place. It is after the fibrosis of the lung has broken down the natural resistance of the physically strong men who enter the industry that invasion with tubercle bacilli takes place. This seems to me to explain the late development of tuberculosis among that class of operatives and emphasizes the industrial origin of the disease.

But aside from statistical data pointing to the industrial origin of tuberculosis in certain industries, it is often quite easy in the individual case to estimate the effect the industrial hazard had in the development of the ill-health.

Here are a few illustrative cases from the Industrial Clinic of the Massachusetts General Hospital:

A man previously well and free from symptoms develops a dry, harassing cough immediately after starting work as a rag-sorter. There are no physical signs in his lungs to account for his cough. Rag-sorting, as you may imagine, is a very dusty work, and it is highly probably that his cough is the direct result of his dust inhalation.

A young man of twenty presents himself at the clinic with signs of advanced pulmonary tuberculosis. His father and two sisters died from tuberculosis. He started to work in a woolen mill as a carder at the age of sixteen and has been during the four years of his work constantly exposed to the dust of the carding machines. It does not seem as if there can be any doubt that the dust exposure, to say the least, accelerated the development of tuberculosis in a man so definitely predisposed to the disease. It seems as if it should have been somebody's job to prevent a young person with such a family history from entering such hazardous work.

A man of forty-three who worked at the carding machine in a woolen mill for twenty-seven years develops tuberculosis. He has no tubercular family history, his habits are good. Is it not highly probable that his dust exposure of many years' duration was the important factor in the development of the disease?

Dust exposure, however, is not the only hazard in industry which is responsible for tuberculosis or non-tuberculous disease of the respiratory organs. Among the hazards other than dust which frequently predispose to respiratory diseases are exposure to extremeness of temperature, more particularly exposures to sudden changes of temperature, as illustrated by the following cases:

A man of twenty-seven complains of frequent attacks of colds, with severe cough. He has lost some weight. His appetite is poor and he has occasional night-sweats. Physical examination is negative except for slightly increased signs in the right apex. This man is engaged in a wholesale fruit house and his job is to load teams with cases of fruit for transporta-

tion. The carrying of the heavy boxes causes him to perspire very freely, while perspiring he has to enter a cellar which contains huge ice-chests and is lined with ice-pipes. This process of getting chilled while perspiring is repeated many times during the working day and causes him to catch cold very frequently. He gave up work for some time on two occasions, during which time he felt much better, but the colds returned on his resumption of the work.

A man of twenty-five complains of pain in his left chest. He has a severe cough and raises a good deal of sputum, which is sometimes streaked with blood. He has frequent night-sweats and he has lost weight. His temperature is 100.4° F., pulse 110. There is diminished resonance in the tops of both lungs, over which areas the breathing is bronchial, with numerous râles. His sputum shows the presence of tubercle bacilli. For eight years this man has been working as a stoker on a steamboat. He is exposed to extreme heat, perspires freely while in the fire-room, and then goes on the deck where it is cold. His bunk is in a dark, damp, cold, and poorly ventilated room.

A woman of twenty-seven complains of frequent colds for the last six years, and a cough which is persistent without remission for the last two years. She raises a good deal of sputum, has night-sweats, and has lost some weight during the last year. The lungs show dulness at the right apex, with prolonged high-pitched expiration. *x*-Ray examination of the chest shows extensive peribronchial thickening.

She has been employed as a cook in a restaurant for ten years. For the last few years she has worked in a small kitchen without any windows in it, the only air coming into the room is from an adjoining dining-room. The room is very hot, she perspires freely at her work, and frequently has to go to the refrigerating room for supplies.

Cases could be easily multiplied where investigations into the patient's occupation revealed the probable cause of tuberculosis or non-tuberculous diseases of the respiratory organs.

Postural strains or localized strains are very often responsible for states of ill-health, as illustrated by the following cases:

A man of twenty-two complains of general weakness, poor appetite, pains in the lumbar region, and also in his right arm and leg. The pains have become so bad that he had to give up his work. Physical examination is entirely negative. This man came to this country from Portugal eleven months ago. He was a baker. He got a job here in a tannery washing skins. He works in a wet cellar. The work is very hard and disagreeable, and he feels that it has completely exhausted him. He was not physically fitted for such laborious work.

A woman of twenty-three complains of pain in her right shoulder and weakness of her right hand. The physical examination is entirely negative, except that the motions of the right shoulder are somewhat limited. This woman is working as a bone-puller in a fish loft; she pulls the side bones from cod-fish. There are 34 side bones in each fish, and she sometimes does as many as 130 fish an hour. It requires considerable pull to tear out the bones; she is engaged in this work for about five hours every day. The room where she works is very cold, and the fish is often so cold as to make her hands very numb. This case well illustrates the localized strain effects so often met with in industry.

In addition to these industrial hazards which are more or less inevitable in particular industries, though in many instances more adequate protection could be provided, there are conditions inimical to health found in industries which have no relation to the industry itself. I refer to the unsanitary conditions characteristic of certain industries. The unclean, poorly lighted, poorly ventilated workroom gives rise to no one disease, but reduces the resistance of the worker and keeps him in a state of impoverished health.

I have so far referred to the two main groups of diseased conditions arising from industry: (1) The specific industrial diseases, and (2) the diseases in which the industry acts as a predisposing cause rather than the immediate cause. There is still, however, another field about which we know little or nothing as yet. I refer to the general fatigue effects from the stress and strain of modern industry.

We unfortunately have no methods of measuring or estimating fatigue. We will have to learn a great deal more about the chemistry and the physiology of fatigue before we shall be able to demonstrate the baneful effect of chronic fatigue on the organism. But I firmly believe when the chapter on industrial fatigue is finally written that it will be found to play no small rôle in the causation of the so-called degenerative diseases of middle life.

At present our estimate of fatigue can be based only on observation of the quality and quantity of the output of the worker, and on the rate of the occurrence of accidents in industry. It is quite clear that the fatigued worker is not able to make as quick or as proper adjustments of his movements, or, as Ash demonstrated, the first symptoms of fatigue are an interference with the function of control over all kinds of motor expression, hence fatigue brings about an increase in the accident rate.

Some years ago I analyzed accidents that occurred in a large metal manufacturing plant for a period of ten years; there were several thousands of them. My analysis showed that the number of accidents increased every hour during the morning, reaching the maximum during the fourth hour and, curiously enough, declining appreciably during the last hour of the forenoon. I found the same to be true of the afternoon hours. That is to say, more accidents occurred between 10 and 11 than between 11 and 12 A. M., and more between 4 and 5 than between 5 and 6 P. M. This was to me a rather unexpected development, as one would naturally expect the greatest number of accidents to occur during the last hour of the morning or the last hour of the afternoon when, presumably, fatigue would be at its highest. This paradoxic discrepancy in the accident curve could not be explained by any change in the work or working force during the last hours of the morning and the afternoon periods.

In looking up later some statistics of accidents in European countries I found, curiously enough, the same conditions existed relative to the diminution of accidents during the last hour of the morning and the last hour of the afternoon.

This apparently paradoxic situation was found to be common to a great variety of industries carried on under different conditions. One is therefore forced to assume that this phenomenon must depend upon some fundamental factor in the economy of the organism. What this fundamental factor is still remains to be demonstrated.

Various explanations have been offered for the diminution of the accident rate during the last hours of the morning and afternoon working periods. None of the explanations thus far have been very satisfactory.

May it not be that this phenomenon is due to the fact that during the last hours of the working period the stage of physiologic fatigue has been passed, that all the energy available for the daily work has been exhausted, and that to meet the persistent demand the worker has to draw upon his reserve or potential energy which nature intended for use only in emergencies?

The existing fatigue, which is nature's call for a period of rest to replenish the exhausted energy, disappears when the stores of reserve energy are made available and the impairment of motor control, which is the first symptom of fatigue and which is responsible for the increase of accidents, disappears and the accident rate falls. This constant drain upon the reserve energy is comparable to the state of a person who lives above his income and has to draw on his capital. This constant use of reserve energy impoverishes the organism of the workers and keeps them in a state bordering on exhaustion, so that they are unable to withstand any emergencies, such as an attack by disease-producing forces. Whether this explanation is true or not it seems to me that there is a wide field of most useful and profitable research.

To summarize briefly: Industrial medicine in its wider meaning is a field primarily not for the industrial physician, but for the physician in the general practice of medicine. He must recognize that states of ill-health are in many instances due to the hazards of industry. Such recognition will often enable him to make an accurate diagnosis in an otherwise obscure

case, and through the aid of the health authorities often prevent the occurrence of other cases.

The field of industrial medicine is not only a profitable one for the clinician, but is a fruitful field for the research worker, for our knowledge in this important field is very small, and the problems that await solution are many and pressing.

CLINIC OF DR. LEWIS WEBB HILL

CHILDREN'S HOSPITAL

NEPHRITIS IN CHILDREN

Presentation of 3 Cases, and Discussion of the Differences Between Nephritis in the Child and in the Adult

February 17, 1919.

I WISH to present today 3 cases of nephritis, and to discuss with you some of the differences between nephritis in the child and in the adult. These cases represent by no means *all* the types of nephritis that we see in childhood, but they are of especial interest because they differ considerably in many respects from cases of nephritis in older persons.

CASE I.—Here is a boy, eight years old. He looks well, as you can see, save perhaps for slight anemia, and rather dark circles under his eyes. His *family history* is unimportant except for the interesting fact that his younger brother at home now is suffering from the same condition that he has himself. His brother was taken sick about a week after he was.

His *past history* is as follows: He has always been a strong, healthy boy, and has never had any of the contagious diseases except measles, which he had two years ago. His only illness has been tonsillitis, and during the last three years he has had two attacks which have lasted usually about five days. A year ago, after an attack, he suffered from swollen glands in the neck, which, however, disappeared in a few weeks.

His *present illness* began two weeks ago. At this time he had tonsillitis. The tonsils were much enlarged, showed white spots, and there was considerable prostration and fever. He stayed in bed three or four days with this, the throat cleared up, and he got out of bed feeling well. A day or two later his mother

noticed that his urine was bloody and that his face was a little puffy, so brought him to the hospital for treatment.

Physical examination shows a rather thin, pale boy, who does not seem very sick. The tonsils are not at present large, but are rather lobulated, inflamed, and unhealthy looking, with large crypts. His teeth are good. On each side of the neck there are two or three small, hard, non-tender lymph-nodes, the size of marbles. The heart, lungs, and abdomen are negative. There is no edema anywhere. Here is a specimen of his urine, and I think you will all agree that it is unusually bloody, even for a case of acute nephritis; it looks almost like clear blood, and there is a heavy dark red sediment in the bottom of the tube. It contains a large trace of albumin, and microscopically shows large numbers of red blood-cells, many small round cells, a few pus-cells, and a very rare granular cast. The phthalein test is 55 per cent. The blood urea nitrogen is 14 mgm. per 100 c.c. blood (normal).

The two-hour renal test (see Case II) shows a decided fixation of specific gravity, at a low level, as follows:

Time.	Specific gravity.
6–10	1015
10–12	1016
12–2	1015
2–4	
4–6	1015
6–6	1014

At no time has there been any edema, and on a daily fluid intake of 48 ounces he passed usually from 28 to 38 ounces of urine.

His systolic blood-pressure is 98, the diastolic 60.

Discussion.—This boy, I think you will all agree, does not present the picture of our ordinary text-book conception of acute nephritis. His urine secretion is good, he has no edema, the blood-pressure is normal, he does not seem sick, and his urine contains an extraordinarily large amount of blood, but very few casts.

It is a streptococcus condition, probably a glomerulonephritis, and is due possibly to damage of the glomerular vessels by

the streptococcus toxin, or perhaps to their actual invasion and occlusion by the streptococcus itself. It is seen in adults, but is not, I should say, the ordinary type of nephritis from which they suffer. It is, however, the most common type of nephritis that we see in children and is almost always secondary to tonsillitis. It should be called "acute hemorrhagic nephritis," and its distinguishing features are lack of marked edema and a very bloody urine containing *very few casts*. Indeed, I have seen physicians who were not familiar with children doubting the occurrence of nephritis at all, on account of the scarcity of casts, and searching for some other condition which would cause hematuria, such as renal stone. The importance of tonsillitis as a cause of nephritis is not usually recognized, but it is, in children, at any rate, a much more common cause than all the other infections combined, not excluding scarlet fever. The nephritis starts usually not during the tonsillitis, but anywhere from a few days to two weeks after the acute throat symptoms have subsided. It lasts in most cases from four to twelve weeks, but occasionally small amounts of albumin and of blood may persist in the urine for many months. A few patients ultimately develop chronic nephritis, from which they finally die, but it is rare for a fatal outcome to occur during the acute stage. Functional tests are moderately diminished, but rarely to the extent that they would be in the ordinary text-book type of acute nephritis in adults. The phthalein test in this case would be normal for an adult, but low for a child, the lowest normal limit for children being about 60 per cent. The two-hour renal test shows an abnormal response; normally there should be a wide difference in specific gravity between the two-hour specimens. The blood urea is normal. In this particular case the functional tests are interesting, but not particularly valuable, as we know from the boy's general condition that his kidney function cannot be much diminished. In fact, as far as treatment goes, we could handle this patient exactly as well if we had not done the functional tests. In chronic nephritis they may be of considerable value, as we shall see in the next case.

How shall we treat this boy? In the first place, he should

be kept quiet and warm. He should not get out of bed for anything, and should be wrapped in an extra blanket. Exertion and cold are two of the worst things a child with acute nephritis can be subjected to. He should have one good bowel movement a day, but there is no necessity for purging him, as his urine secretion is good, and he has no edema. In these hemorrhagic cases without edema it is well to give as much fluid (within reason) as the patient can handle, as it is probably of value to flush the kidneys and aid in diluting and in eliminating toxins. This boy has been taking 48 ounces of fluid daily, as you can see from his chart, and has handled it well, as his urine secretion is about what it should be, and he has not gained in weight. The diet is important, and here again we need to restrict him very little. There is no edema; therefore there is no good reason for giving him a salt-free diet, although we should be careful, on the other hand, not to let him have an excessive amount of salt. The greatest prinicple in treating any case of acute nephritis is to spare the kidneys as much as possible. Although it is possible that this boy's kidneys might be able to handle a large amount of protein waste, it is only reasonable to cut his protein allowance to the minimum amount which will cover the nitrogenous needs of his body. We have no very clear idea what the nitrogenous needs of young children are, but we know that they are greater than those of adults, and if we give this boy about 2 gm. of protein for each kilogram of body weight we shall probably keep him in nitrogenous equilibrium. You will see from his chart that in the hospital we have the nurses calculate carefully the total calories and the protein of his diet, but in private practice this is not necessary. If, during the acute stage, while the urine contains considerable blood, meat, eggs, and meat soups are omitted, and the diet is made up of milk, cream, butter, cereals, vegetables, and fruits, we shall come very close to what is desired. The only drug that is of any value is iron, to compensate as much as is possible for the large amount of blood that is lost daily in the urine, and this boy has been having 5 grains of the saccharated oxid of iron three times a day, which is perhaps the best form of iron to use with children.

We shall begin to think of letting him up when his urine contains only a rare red blood-cell microscopically, and no albumin, or the slightest possible trace. If considerably more blood appears in the urine after he has been up, and persists, he will have to go back to bed again. Many of these cases may show a very small amount of albumin for a considerable time, and it may not mean much, but if the blood persists, even microscopically, the patient should be treated very carefully, and should be kept in bed most of the time.

When is the best time to take out the tonsils? Some observers advocate doing this immediately, and some say to wait until the nephritis is entirely recovered from. Personally, I believe a middle course is best, and should advise tonsillectomy for this boy as soon as the acute stage has passed and his urine contains only a moderate amount of blood.

CASE II.—The next case presents a somewhat different problem. This girl of ten was brought to the out-patient department of the hospital with the following history:

Family history unimportant.

Past History.—Measles at one year, diphtheria at seven years, no scarlet fever, pneumonia, rheumatism, chorea, or tonsillitis. She has never been a very strong child, but has been apparently well up to the onset of her present symptoms.

Present Illness.—For the last year she has been pale and weak, and gets tired very easily. Her appetite is poor, and she has gained only a pound in a year. No cough. Has slight dyspnea and palpitation on exertion. No precordial pain. No nocturia, polyuria, dysuria, or hematuria. No excessive thirst, no enuresis.

Physical Examination.—Shows a rather thin, washed-out looking girl, rather tall for her age, and standing with a marked lordosis. Her head is negative. The eyes are normal. The nose is negative. The tonsils are not large and are apparently normal. The teeth are poor, and a definite pus-pocket can be seen on the gum at the root of one of the lower premolars. The tongue is heavily coated, and the breath, though not very foul, is rather unpleasant. There is no general glandular enlarge-

ment. The heart measures 2.5 x 8 cm., the sounds are of good quality, and at the apex there is heard a short, soft, and rather insignificant systolic murmur, which has only a very narrow area of transmission ("functional"). The pulmonic second sound is greater than the aortic second. The lungs are negative. The abdomen and extremities are negative. There is no edema.

Diagnosis.—We see many such children in the out-patient department, and the vast majority of them with the symptoms that this girl complains of are suffering from no definite disease, but simply from the wrong sort of food, hygiene, and home conditions. We have not completed our examination in such a case as this, however, until we have looked at the urine. The urine is clear, with a specific gravity of 1022. Contains a slight trace of albumin, no sugar, and has an occasional hyaline and granular cast in the sediment, with a few blood-cells. Here we have, then, probably either a mild chronic or subacute nephritis, or possibly postural albuminuria, and we have found a definite cause for her symptoms. She was sent into the wards for study. If she has a postural albuminuria the urine should be free from albumin while she is kept in bed, and should contain albumin when she is up and around, particularly if she is up and the lower part of her body is immobilized. The general appearance of the patient and her very evident lordosis would suggest this. She was put to bed, and several urine examinations were done while she was in bed at different times, and while she was up and around, with the following results:

Specific gravity.	Albumin.	Sediment.
Up—1009 (quiet).	S. T.	Rare hyaline cast, and a few red blood-cells.
In bed 1—10015.	S. P. T.	No red blood-cells or casts.
In bed 2—1018.	S. T.	Rare granular cast.
Up and moving—1022.	L. T.	Rarely a granular cast.
In bed—1018.	V. S. T.	Few red blood-cells and casts.

It would seem that she has, therefore, not a postural albuminuria, but a chronic nephritis, as staying in bed did not render the urine albumin free.

Discussion.—My reason for showing this child is to call especial attention to the fact that many cases of chronic neph-

ritis in children may be of a very mild character, and present a totally different picture from chronic nephritis in the adult. We see in children many times the "chronic parenchymatous" nephritis of the adult, with the severe anemia, extensive edema, etc., and usually can suspect the diagnosis from the appearance of the patient, but there is nothing in this type of case to suggest nephritis until we examine the urine. Occasionally, if these children happen to pick up an acute infection of any sort, they show an exacerbation of the nephritic condition, but they rarely have much edema or seem very sick. The blood-pressure is not elevated (indeed, it is likely to be lower than normal) nor is the heart hypertrophied. The urine ordinarily shows only a small amount of albumin, with a rather insignificant sediment, in which a few casts of the granular and hyaline varieties and an occasional blood-cell may be seen. There is always the necessity, in these mild cases, of differentiating the condition from postural albuminuria, and this can be done, as it was in this case, by examining the urine for albumin a number of times while the patient is in bed, and again while he is up and around. In postural albuminuria the second and succeeding specimens passed after the patient has been put to bed should always be albumin free, and we cannot make a diagnosis of postural albuminuria unless this condition holds. So far as I know, there is no condition in the adult which corresponds to this type of nephritis. You will note particularly that there is no hypertrophy of the heart, and that the blood-pressure is not elevated; occurrences which help us a good deal in the diagnosis of chronic nephritis in the adult. In the child these signs may occur, and are of value in diagnosis if they do, but are more often absent than present in this mild type of case. In this particular case the etiology is obscure; there is no history of a preceding acute nephritis, although it is possible that her kidneys may have suffered injury during her diphtheria three years ago, or that the abscess that she has now at the root of one of her teeth may have something to do with it. The tonsils appear harmless, but may contain pus-pockets.

What we wish to know particularly about this girl is the

extent of damage that has been done to her kidneys. From her general appearance we should infer that they have been little damaged, but the functional tests may help us in deciding this, and it is in just this type of case that they are sometimes of value. Here again we must contrast the adult and the child. In the adult high or normal functional tests in a case of suspected chronic nephritis would almost certainly indicate to us that the kidneys were little damaged. In the child this is not necessarily true, and I have seen several severe chronic cases with normal phthalein tests and normal figures for blood urea. The "two-hour renal" test, as elaborated by Mosenthal, which I have used with children in a somewhat modified form, is more reliable, and I have seen few cases of well-established chronic nephritis which did not show deviations from the normal. Low phthalein excretion, high blood urea, and abnormal two-hour tests give very reliable evidence, on the other hand, that the kidneys are severely damaged and that the prognosis is poor.

This girl had a phthalein of 65 per cent., which is normal, but does not help us much one way or the other. The "two-hour" test is valuable and extremely simple. The child is put on a full diet containing plenty of meat, with 10 ounces of fluid at each meal. She is also given 2 grains of caffein sodium benzoate and about a gram of salt with each meal. For the sake of uniformity, in the wards we use the following diet, but it is not at all necessary that the child take all of it, or that it be measured carefully or weighed. It is essential that no fluid or food be taken between meals, and that the same amount of fluid is taken at each meal.

Breakfast:	*Dinner:*
Cereal, 2 tablespoonfuls.	Chopped meat, 2 tablespoonfuls.
Bread, 1 slice.	1 egg.
Butter, ½ square.	1 potato.
Apple sauce, 2 tablespoonfuls.	Butter, 1½ cubes.
Milk, 6 ounces.	Milk, 6 ounces.
Water, 4 ounces.	Water, 4 ounces.
Extra salt, 1 gram.	Extra salt, 1 gram.
Caffein sod. benzoate, 2 grains.	Caffein sod. benzoate, 3 grains.
Supper:	
Same as breakfast.	

The urine is collected at two-hour intervals, and the specific gravity of each specimen is taken. Normally there should be a variation in gravity of the different specimens of 8 points or over between the highest and the lowest. This indicates that the kidneys have the power of varying the concentration of the urine according to the demands made upon them. In damaged kidneys this power is lost, and there is very little difference in gravity between the two-hour specimens. Sometimes all the specimens may show almost exactly the same specific gravity. this girl had a normal two-hour test, as follows:

Time.	Specific gravity.
6–8	1019
8–10	1006
10–12	1020
12–2	1021
2–4	1028
4–6	none
6–6	1022

As criteria of the efficiency of this particular girl's kidneys we can include:

1. Her general condition.
2. The character of her urine.
3. The two-hour renal test.

Her general condition is certainly not compatible with a severe kidney damage. Her urine shows a comparatively small amount of albumin and few casts, and her two-hour test is normal. We may, therefore, say that she has not enough damage to her kidneys at present to materially interfere with their functional efficiency.

Just what will happen to this girl? Will she rapidly develop a diffuse severe chronic nephritis and finally die from it? Will she slowly develop a contracted kidney, and have ten or fifteen years from now what is called "chronic interstitial nephritis," or will she finally get over her nephritis entirely?

The first possibility is, I think, unlikely. It is quite likely that a good many of the cases of chronic interstitial nephritis that we see in after-life may have had their foundation on some

such basis as this, and it is possible that this child may slowly develop a contracted kidney. On the other hand, it is not at all unlikely that her albuminuria will gradually disappear, and in a few years she may have a perfectly normal urine.

How should she be treated? The two most important things for her to remember are to avoid infection and overexertion. Her teeth should be put in order by a competent dentist and her tonsils should probably be removed. She should avoid chilling and exposure. She should not go in bathing, she should engage in no violent sports. These are the main things to bear in mind; the diet in such a case as this is entirely secondary. It would be quite wrong to put this girl on a closely restricted diet —she should have meat once a day, she need not particularly avoid salt, and for the rest of it may have about what would be a rational, normal diet for any girl of her age. I should give her 5 grains of the saccharated oxid of iron thrice daily, and also the following prescription which we use a great deal here at the hospital:

℞. Nux vomica..... ℥ij
Glycerinated elixir of gentian..... ℥vj
Sig.—One teaspoonful before each meal.

Case III.—This boy of seven represents a very rare condition in childhood—*chronic interstitial nephritis*, which merits a brief discussion on account of its rarity.

The *family history* is not remarkable. His father and mother are both apparently in good health. He is an only child and there have been no miscarriages.

Past History.—He has never been a robust child, and has always been somewhat thin and undersized, but has been considered healthy by his parents except for the "attacks" for which he enters the hospital. He has had no acute infectious diseases.

Present Illness.—Since he was a year old he has been subject to "bilious attacks," which have been becoming more frequent in the last two years. Previous to two years ago one would occur perhaps every month or two. In August, 1916, he began to have them once a week; for the last few month he has had an average of two attacks a week. The attack usually starts

at night. He wakes up at 3 or 4 A. M. with headache, crying with the pain. After an hour or two he begins to vomit. The attacks usually last from twelve to twenty-four hours, after which he is fairly comfortable, with the exception of an occasional moderate headache, until the next one. The attacks seem to bear more relation to overfatigue than to food. He is very thirsty always—drinks a large amount of water and passes a great deal. Dr. S., the family physician, has examined the urine three or four times during the past year, and has found it normal at every examination, until a few days ago it was found to contain $\frac{1}{10}$ per cent. albumin and a few pus-cells in the sediment. It contained no casts or blood. For the last two months his headaches have been nearly continuous and of extreme severity, so severe that at another hospital his head was *x*-rayed with the possibility of a pituitary tumor in view. Two months ago on account of poor vision he was taken to an eminent oculist, who examined his eyes and said that there were one or two small spots on the right retina near the disk. He prescribed glasses, but made no diagnosis of any cerebral or severe ocular condition. The glasses did not help him at all. The "bilious attacks" have been so frequent for the last month that he has been in bed with them most of the time. A week ago, according to his family physician, there was a good deal of blood in his urine. Four days ago he was taken to the same oculist he went to before, who reported a diffuse retinitis of both eyes, which he said looked albuminuric. The next day he entered the hospital for study.

Physical examination shows a pale, very much underdeveloped boy, weighing only 37 pounds. The pupils are equal, regular, and react to light. Both retinæ show a diffuse retinitis. The tonsils are small and appear normal. The heart is slightly enlarged, but there are no murmurs. The aortic second sound is decidedly louder than the pulmonic second and is definitely accentuated. The lungs are negative, as is the abdomen, and the liver and spleen are not felt. The genitalia are small and much underdeveloped. The knee-jerks are equal and active. There is no edema. The urine has been examined several times, and has always been of low gravity (1010 to 1014), has con-

tained very slight traces of albumin, and at most examinations rare granular casts and a few blood-cells. The phthalein test is 55 per cent. (definitely low for a child). The systolic blood-pressure is 210, the diastolic 120.

Discussion.—This condition is apparently the same thing as "chronic interstitial nephritis" in the adult. It is very rare in children, and not more than 20 or 30 cases have been reported in the literature. The etiology is very obscure, and in some instances seems to be of a familial tendency, as there are cases on record where two or more children in the same family had the condition. Syphilis probably is not an etiologic factor. The most striking and interesting characteristic of the condition is the occurrence of infantilism, and nearly all of these children are very much underdeveloped both mentally and physically. The symptoms are those of chronic interstitial nephritis in the adult. The blood-pressure is usually very high, the heart is hypertrophied, headaches, visual and gastric disturbances are common, and there is polyuria and polydipsia. The urine is of constant low gravity, contains a small amount of albumin, and a rare cast or blood-cell in the sediment. The urinary changes, aside from the low gravity, are often so slight as to pass unnoticed, and it is possible (as in this case) to make several urine examinations, and to find nothing abnormal save the low gravity. The disease is so rare that if one does not have it in mind it may be overlooked. The blood-pressure is the best single guide, and it is always advisable to take blood-pressure readings on any underdeveloped child with headache or visual symptoms. At autopsy the kidneys are very much shrunken in size, and histologically show the typical changes of "chronic interstitial nephritis. This is the only form of nephritis in early childhood in which the blood-pressure is constantly high, and in which the cardiac hypertrophy is enough to be of any diagnostic value. In other types of nephritis in children, both acute and chronic, the blood-pressure may be high or normal, and is, therefore, not of the same diagnostic value that it is in the adult.

The prognosis of this condition in children is always bad, and the chances are that this boy will live only a few months at best.

CLINIC OF DR. FRANKLIN W. WHITE

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IMPROVEMENT IN THE MEDICAL TREATMENT OF CHRONIC ULCER OF THE STOMACH AND DUODENUM

General Principles of Treatment.—We will first outline the principles of treatment and then give the details of our routine. We are always somewhat at a loss in treating a disease of whose exact cause we are not sure. The latest trend of opinion favors an infectious origin. Rosenow and others have produced ulcer by bacterial infection, and we now try to root out all sources of infection in ulcer cases. We clean up pus-pockets about the teeth and tonsils and sinuses, as well as remove an infected appendix or gall-bladder. This is good for ulcer patients and for anyone else, and we have been very scrupulous about it for the past few years. It is too soon to judge end-results. Our impression is that recurrences are less common where this is done.

Whatever the origin of ulcer, whether infectious or not, we want to know *what keeps it from healing and makes it chronic.* This has been well established both by experimental work and observation of patients, and serves as a working basis for treatment.

The most important factor which retards the healing of ulcer is delay in emptying the stomach. A second factor is the presence of an abundance of free acid. A third factor is mechanical, the irritation of the ulcer by peristalsis, spasm, and food particles. Therefore in order to heal a chronic ulcer we must avoid everything which increases gastric acidity, peristalsis or spasm, or the total work of the stomach.

We must use bland foods which cause least acidity, least

secretion, least peristalsis, and leave the stomach quickly, and use other measures which reduce secretion, peristalsis and spasm, and promote emptying of the stomach (sedatives, atropin, lavage, etc.).

We must especially relieve pyloric spasm, which is a far more common cause of retention of food than tissue narrowing.

There is no question that psychic influences, nervous shock, mental strain, and overexertion play an important part in developing and continuing ulcer. This is no doubt brought about by increased spasm and increased secretion of gastric juice. We must rest and build up our patient and teach him to avoid emotional excess and try to change his faulty mental habits.

Above all, we must *individualize the treatment;* all ulcer cases can no more take the same treatment than all diabetics can eat the same diet. We must treat the ulcer according to its position and size and the changes in function which go with it. The treatment is an experiment to be watched and followed, like increasing the diet in a diabetic.

We can be more patient with the *duodenal* than with the gastric ulcer, and continue the treatment longer in the face of difficulties, for we have not the fear of cancer hanging over us.

We can afford to try medical treatment in the *small gastric ulcer*, while with the large one we prefer to call a surgeon. Some men consider the demonstration of *any* ulcer in the stomach as an indication for a surgeon. We do not agree. If the surgeon tries to excise every ulcer of the stomach regardless of size or location, more patients will die of operation than of cancer.

The *ulcer with retention of food* needs special attention. It is on the borderline between medicine and surgery. We must relax spasm of the pylorus by the free use of atropin, or, if need be, remove the contents of the stomach at bedtime with the tube, and do everything possible, including rest in bed, hot compresses, and alkalies, to reduce irritation, infiltration, and edema about the ulcer. Here we must get prompt and definite good results from medical treatment, otherwise the case is surgical.

Our only excuse for using medical treatment in these cases

is the fact stated by Dr. W. H. Mayo years ago, that *delay in emptying the stomach in chronic ulcer is due to actual tissue narrowing in less than 10 per cent. of the cases.* It is very important to decide whether delay in emptying the stomach is due merely to spasm of the pylorus or to actual tissue narrowing. I want to emphasize the use of atropin in deciding this question. Many a case of obstruction, which we believe at first to be organic, relaxes almost entirely under the use of atropin and other treatment. This is well illustrated by our first patient.

CASE I.—Mr. R. C. P. C., age sixty-nine; a very hard-working engineer, has had a mild diabetes for ten years, easily controlled by diet. He had attacks of indigestion in 1900, 1906, 1912, 1918, lasting a few weeks to several months, with intervals of good health between. During his recent attack, which has lasted a month and which is like the previous ones, he has had epigastric distress or pain chiefly between meals, with heartburn and acid regurgitation. There has been no nausea and no vomiting. He has used the usual diabetic diet of eggs, meat, fish, and some green vegetables and fruits, with a small amount of bread and cereals. He has had a blood-pressure of 190 to 200 for about five years, with rare headaches and dizziness. His average weight has been 148 pounds up to three months ago, and he now weighs 133 pounds. He is a well-preserved old man; with good teeth, his tongue is fissured; the heart is somewhat enlarged. There is nothing to be found in the ordinary examination of his abdomen. His urine shows a slight trace of albumin and 1 per cent. of sugar. His stomach, after a test-breakfast, showed low normal secretion, Free HCl 20; total acid 39; no mucus; no blood with benzidin. The amount removed from his stomach after a test-breakfast was 329 c.c., showing that very little of the meal had passed the pylorus.

The Roentgen examination showed a stomach of smooth outline, with active peristalsis; a deformed duodenal cap which I was never able to fill out even under a long fluoroscopic examination and series of plates. Very little of the barium meal had passed out of the stomach at the end of six hours, and at

the end of twelve hours fully half of it still remained in the stomach.

The diagnosis of duodenal or pyloric ulcer was made, and in spite of the very marked obstruction medical treatment was begun—such as rest in bed, hourly feedings, alkalies, and atropin—with the hope that a great deal of the obstruction was due to spasm and inflammatory edema and would promptly subside. I thought the possibility of cancer was remote, but the stools were tested every few days for blood. They were found negative after the first four or five days.

Under this medical treatment he made an uninterrupted recovery, gained about 15 pounds in weight, and has had absolutely no gastric symptoms for a year.

This case shows how marked an obstruction may be present as a result of secondary spasm of the pylorus associated with an ulcer nearby, and how completely this type of obstruction may sometimes be relieved by appropriate medical treatment.

The patients with a *good-sized gastric ulcer*, with a crater as wide as a 5-cent piece, well up on the lesser curvature, who refuse operation, are a trying class. Medical treatment sometimes gives excellent results, but we always feel as though we were playing with fire, and the patient may remain only partially well. (See Case V.)

In the *ulcer which bleeds* everything must be sacrificed to stop the bleeding, and the case overhauled and classified later.

Last, but not least, people differ greatly in their general make-up and ability to tolerate an ulcer. The patient must be treated as well as the ulcer.

Hospital patients often require surgical treatment of ulcer when private patients would not, because the former are unable to favor themselves by rest, good hygiene, and a carefully selected diet for a long period.

Follow-up System.—I want especially to emphasize the *need of a good follow-up system* in chronic ulcer. This is just as necessary as in diabetes. These ulcers are essentially chronic. This should be carefully explained to the patient. The greatest failure I see in medical treatment is the lack of following the

cases carefully for months and protecting the stomach from irritation for a long time.

Every ulcer is different from the last, and every medical treatment is in the nature of an experiment, and needs careful watching and frequent modification. There is no such thing as "the ulcer treatment" any more than "the diabetic diet." We do not want to lose track of a case of diabetes and only have him report when there is sugar in the urine, neither do we wish to lose track of a chronic ulcer and only have him report when his symptoms are severe. To watch the patient with chronic ulcer and protect him from recurrence is just as much our business as to keep the diabetic sugar free.

We do not depend simply on the patient's symptoms and general condition, we use the Roentgen ray freely to *watch the anatomic changes in the ulcer and their effect on gastric function.* In our follow-up system we emphasize the following points: Blood tests of the feces are made every few days at first until the stools are persistently negative. After convalescence, in addition to regular reports at intervals on general condition, symptoms, weight, etc., we make a Roentgen-ray examination of the patient at the end of three or four weeks, and at intervals of three or four months for a year or more after this, depending on the case, in order to study the anatomy of the ulcer, changes in size, peristalsis, persistent spasm, the emptying of the stomach, etc.

Of the occasional functional tests, the motor test is the most important. We use the fluoroscope personally because we usually combine the motor test with the observation of the ulcer itself. The general practitioner will naturally use the stomach-tube for the motor test. It is, of course, much simpler and more readily available. This is not the place to discuss the relative value of barium meals and ordinary meals in testing the motor power of the stomach. They run closely parallel, as a rule.

The response of the patient to treatment as shown by symptoms, motor tests, blood tests, and the anatomy of the ulcer soon gives us a clue to the expectation of cure. This observation of the patient has great value in protecting him from

cancer. It is safe to say that in nine-tenths of the ulcers which "go bad" cancer could be avoided by an efficient follow-up system and constant weeding out of cases unsuitable for medical treatment. Not a single unpleasant surprise of this kind has occurred in our ulcer cases since this follow-up system has been carried out.

A follow-up system is equally valuable in cases after operation. In addition to judging the case by general condition and symptoms, we can decide by Roentgen examination about the anatomy of the ulcer, whether or not the new opening is working well, if it is in the right place, or too large or too small, whether or not food goes freely and promptly through it, whether the stomach empties too fast or too slowly, whether the cutting of circular fibers has hurt the muscular action of the stomach and caused food to pile up in the lower end of the stomach below the opening, and whether adhesions are present. More than one examination is usually needed when stasis, spasm, or pocketing is found. These changes are often variable and may entirely disappear after the use of atropin. Neglect of this rule has led to wrong opinions about the stomach and its functions and sometimes to needless secondary operation.

We must have all this data to decide what later treatment is best for the individual case. We have frequently been able to quiet the fears of the patient, lest something had gone wrong with the mechanism of the stomach after operation by demonstrating normal, smooth emptying, and then have stopped the symptoms of irritation by simple medical treatment.

This *medical treatment of ulcer cases after operation* is very important. The surgeon frequently tells his patient after operation that he is now perfectly well and can do anything he likes. I have met this statement again and again, and it is rarely true. The ulcer case with gastro-enterostomy or excision is "damaged goods," and frequently needs some medical advice in order to keep well. I have a long and steadily lengthening list of ulcer patients who need help *after* operation, usually after gastro-enterostomy; not in the first week or few months after operation, but six months or more later. The old symptoms return—

acidity, distress, pain, vomiting—and the patient fears another ulcer, perhaps another operation. Examination almost invariably shows that the patient has become careless in various ways in regard to diet, mental strain, tobacco, or alcohol. Frequently the examination shows a tendency to hyperacidity and spasm and active peristalsis. The stomach is usually empty in the normal time. We have an irritated stomach, not another ulcer. A short course of ambulatory medical treatment has almost always checked these symptoms and brought about a return to health which lasts as long as the patient uses care. I firmly believe that some regular medical advice and follow-up system is desirable in all surgical ulcer cases.

Let me emphasize once more in closing this paragraph on the follow-up system, that chronic ulcer must be considered a serious chronic disease which produces years of individualism and frequently threatens life. It becomes us to take it seriously and *watch the patient carefully for long periods* and neglect no important details which will help him to get well.

It sometimes appears as if there were real *differences of opinion between physicians and surgeons about the treatment of ulcer*. These differences are not real. They are due almost entirely to the difference in the material seen. The earlier and simpler cases come to the physician, the later, more severe and complex cases, are selected for the surgeon to see. Sometimes the surgeon is a little intolerant of any medical treatment of chronic ulcer because he only sees the class of cases in which medical treatment has failed.

Medical treatment, properly used with the new and more exact methods we now have for diagnosis and observation, will cure more chronic ulcers than surgery. Not necessarily because it is a better treatment, but because it is always used first. We must choose our case for medical treatment with care and good judgment, and remember that surgical treatment begins where proper medical treatment leaves off. There is always a good-sized group of cases which we cannot permanently cure by medical means, and we owe a great debt to the surgeon for the development of a successful treatment for the difficult cases of chronic ulcer.

The surgeon sometimes twits the physician with the fact that his "cures" are not all cures, some of them are simply remissions.

What are the signs of cure of an ulcer? A long period of freedom from symptoms, say six months or more, the longer the better, combined with normal gastric functions, absence of blood in the stools, and a marked or complete change in the anatomy of the ulcer (filled up crater, normal peristalsis, no spasm or hour-glass contraction). When an ulcer reappears after such an event, I think we are justified in calling it a *new* ulcer, not the recurrence of an old one. Ulcers in hospital patients recur much more frequently than in the well to do on account of coarse diet, alcohol, hard work, and unfavorable conditions, and therefore need surgery more often.

The **anatomic results of medical treatment of ulcer,** as judged by the Roentgen examination of the patient, are very interesting. They are most easily studied in the ulcers along the lesser curvature which have a definite crater, perhaps some spastic hour-glass deformity, and also in ulcers of the duodenum where there is a characteristic deformity. We have seen the crater of gastric ulcer along the lesser curvature entirely disappear in several cases after medical treatment, but thus far we have not seen a single markedly deformed duodenal cap which has entirely filled out to its former smooth, plump contour after treatment. Evidently a certain amount of scar tissue or adhesions have persisted which still deform the delicate walls of the duodenum. This is true even in cases which have remained entirely without symptoms for a year or more, and which we considered cured. The deformity of the duodenum has greatly lessened, but *not entirely* disappeared. In ulcers at the pylorus without definite crater and with a good deal of induration and scar tissue the anatomic changes after treatment are not striking.

Even in healed ulcers along the lesser curvature the scar tissue usually gives a small, smooth, rigid area over which peristaltic waves do not run.

We must give just a word of warning about using the Roentgen ray to study the anatomy of the healing ulcer. We must be very careful and thorough with the Roentgen examination

and use the fluoroscope as well as take plates for record and for detail. We must not be misled by variations in appearance caused merely by different degrees of filling the stomach and duodenum or different angles at which pictures are taken. It is even possible that food may occasionally stick in the bottom of an ulcer crater and make it appear shallow. Ulcers are usually less well seen after a gastro-enterostomy, as the barium meal may run rapidly out of the new opening, leaving the stomach only partly filled.

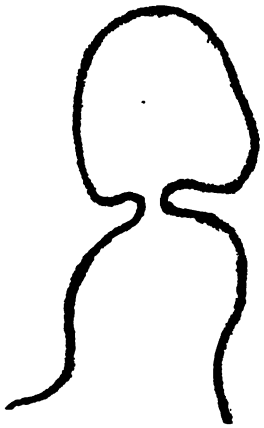

FIG. 242.—Normal duodenal cap for comparison (life size).

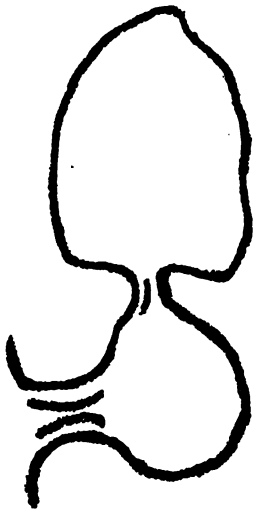

FIG. 243.—Normal duodenal cap for comparison (life size).

These difficulties can be overcome with care. We believe that the Roentgen examination, which has proved so very helpful in diagnosing ulcer, can be equally useful in watching the results of treatment.

The ulcer deformity *in postoperative cases* as viewed by the Roentgen ray behaves in much the way it does after medical treatment. That is, deformity of the duodenum and pyloric region persist to a greater or less degree in ulcer of the duodenum and pylorus, and usually is distinctly less than when first examined. The deformity in ulcers of the lesser curvature

frequently disappears almost entirely in a short time; in others areas of adhesions and scar tissue persist.

Out of a group of about 30 *medical cases* which have thus far been followed for a year or more after medical treatment I have chosen a few to illustrate these points.

CASE II.—Dr. H. A. C.; age forty; in hard general practice; had an infected tooth root and an infected antrum. He has had typical hunger-pains and soda habit, with rare night pain,

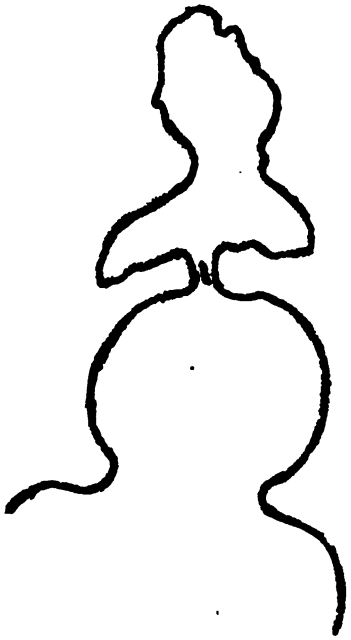

FIG. 244.—Case II. Duodenal ulcer (life size).

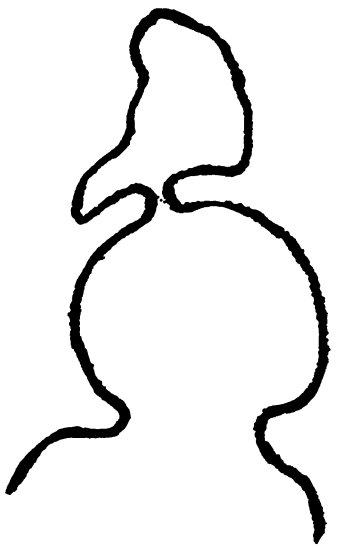

FIG. 245.—Duodenal ulcer, one and a half years later, medical treatment free from symptoms for over a year, some deformity persists (life size).

intermittently, for three or four years. His stomach contents showed very marked hyperacidity, and Roentgen examination showed a deformed duodenal cap as seen in Fig. 244. Compare with Figs. 242 and 243 of normal duodenal caps. The stomach emptied in the normal time.

After taking ambulatory medical treatment of the ulcer he has remained entirely well for a year and a half.

We still have the deformity which is shown in Fig. 245. The

duodenal cap is still very hard to fill, and there is a kink in the second part of the duodenum which remains filled longer than normal, evidently due to adhesions.

CASE III.—Mr. H. K. E. is a business man of forty-six, who had typical hunger-pains for three years. I saw the patient first at the time of acute hemorrhage. A good-sized amount of blood was passed by the bowel and also vomited. A good result was obtained by the usual ulcer treatment in bed.

A Roentgen examination made two months later showed the characteristic deformity of the duodenal ulcer as seen in Fig. 246.

FIG. 246.—Case III. Duodenal ulcer, hemorrhage (life size).

The patient was a very nervous man and had many minor setbacks, but finally became free from digestive symptoms, and has remained so for nearly a year. Figure 247 shows the contour of the duodenum after eight months' absence of digestive symptoms. Instead of the marked deformity of the duodenum found at first with active ulcer, we have left a very small irregularity on one side of the duodenum, evidently a small area of scar tissue.

FIG. 247.—Case III. Duodenal ulcer, one year later, medical treatment free from symptoms for eight months, slight deformity persists (life size).

CASE IV.—Mrs. C. H. is a housewife of sixty-seven, who has had dull epigastric pain, coming two or three hours after meals for a year and a half, with little relief from soda or food. She

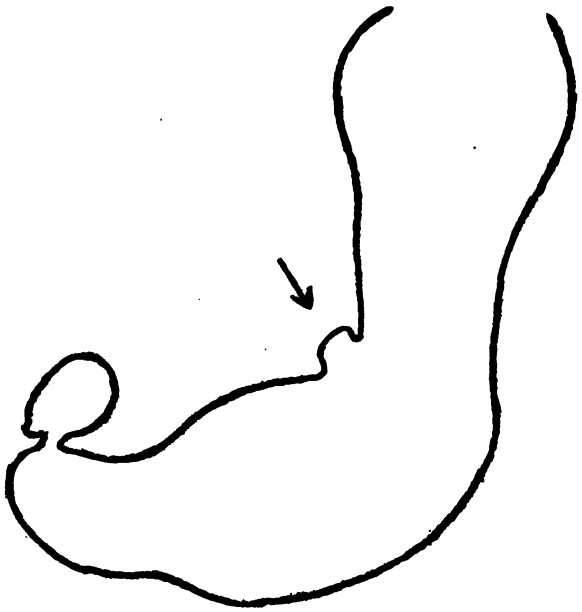

FIG. 248.—Case IV. Gastric ulcer of lesser curvature, operation refused.

has lost 5 pounds in weight. The stomach contents showed low normal secretion, without mucus, with a trace of blood with the benzidin test.

A Roentgen examination showed a stomach which emptied promptly; a characteristic ulcer crater about the middle of the lesser curvature, as seen in Fig. 248. The size of the ulcer crater was a little less than a 5-cent piece.

A diagnosis of gastric ulcer was made, with the possibility of cancerous degeneration and operation advised. The patient

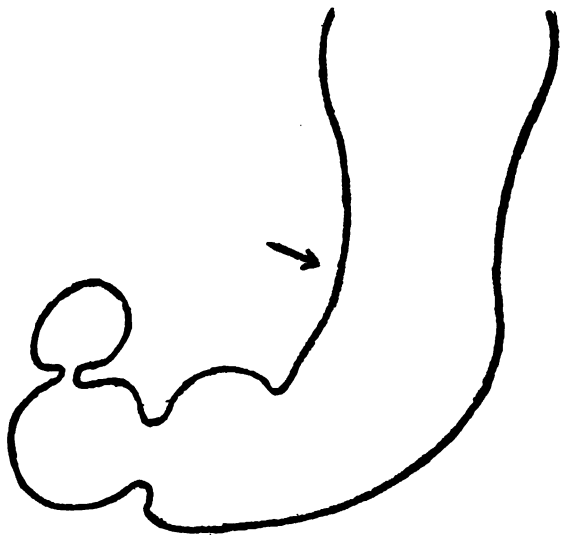

FIG. 249.—Case IV. Six months later, no deformity seen.

refused operation and was put to bed and took the usual ulcer treatment. Her symptoms entirely disappeared. She gained in weight and has remained well for a year and a half.

A Roentgen examination of the stomach six months after beginning of treatment showed no sign of the ulcer at the lesser curvature or any other abnormality of the stomach (Fig. 249).

The next case illustrates a less complete result of medical treatment in a gastric ulcer high up on the lesser curvature.

CASE V.—Mrs. C. P. B., a housewife, aged fifty; formerly well; six months ago began to have sharp epigastric pain with-

out relation to meals, and vomiting soon after meals without blood, and lost 20 pounds in the last six months. Four months ago two cysts of the ovary were removed and a retroverted uterus pulled up into place. Following the operation the stomach behaved well until one month ago, when all the symptoms returned. She is a long, thin, frail looking woman, with a small tender area in the epigastrium. The stomach contents showed normal secretion; a trace of blood with the benzidin test.

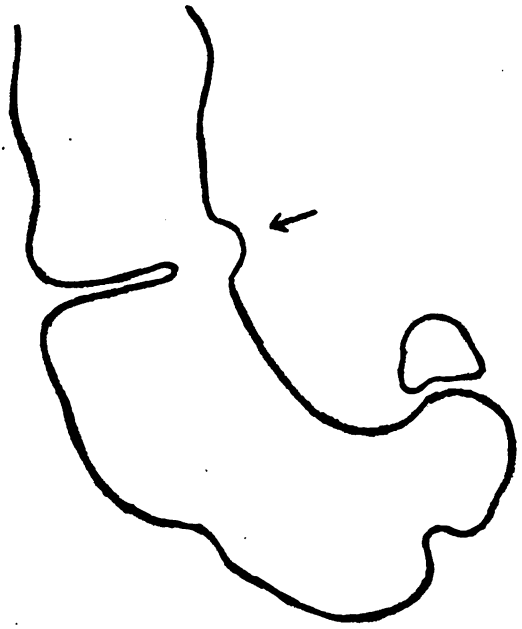

FIG. 250.—Case V. Gastric ulcer of lesser curvature, operation refused.

A Roentgen examination showed a characteristic ulcer deformity about the middle of the lesser curvature of the stomach. The crater of the ulcer was about 2 cm. wide and 1 cm. deep. There was a deep incisura of the greater curvature opposite the ulcer (Fig. 250). There was a good deal of ptosis of the stomach and bowel. The stomach emptied in six hours.

The diagnosis of chronic gastric ulcer was made, and she was put to bed and given the usual ulcer treatment. She gained steadily in every way until at the end of two months she was

free from symptoms and had gained 15 pounds. A Roentgen examination at that time (Fig. 251) showed that the crater of the ulcer is practically filled up smooth. There is a little incisura opposite the site of the ulcer which shows there is still some irritation there. On account of the general improvement and the anatomic change the prognosis was considered to be good. After this time the patient passed out from under my observation.

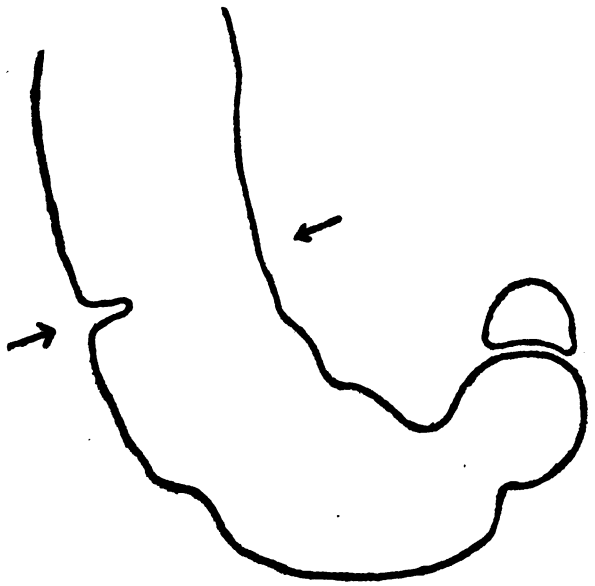

FIG. 251.—Case V. Two months later; no ulcer deformity, small incisura.

In Case VI we have an example of a duodenal ulcer not recovering under medical treatment, but being made fairly comfortable and refusing operation. The anatomic changes in the duodenum remain practically constant for months.

CASE VI.—Mr. A. T. K. is a manufacturer, aged forty; a hard worker; a rather nervous man; now retired for four years; had had many ulcerated teeth, none for a year; for fifteen years has had intermittent attacks of epigastric distress, coming one or two hours after meals, relieved by food or magnesia,

occasional severe vomiting; no blood; no old food; loss of about 8 pounds in weight.

Physical examination showed vague tenderness in the epigastrium; a moderate increase in gastric secretion, without blood or mucus.

Roentgen examination showed a stomach normal in position and outline, with deep vigorous peristalsis. There is a marked constant irregularity of the first portion of the duodenum

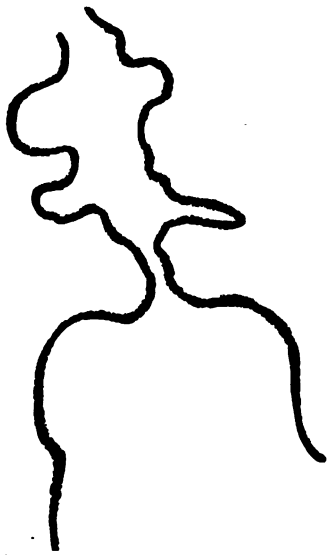

FIG. 252.—Case VI. Duodenal ulcer (life size).

FIG. 253.—Case VI. Six months later, medical treatment with partial relief of symptoms, operation refused, deformity persists (life size).

(Fig. 252). The appendix was seen, apparently normal. Examination of the gall-bladder was negative. A diagnosis of duodenal ulcer was made.

He was put on ambulatory ulcer treatment, with great relief of symptoms. There is a tendency for a slight disturbance to recur with indiscretion in diet, and it has been impossible to entirely clear up his disagreeable symptoms. He had gained 5 pounds in weight at the end of six months.

The second Roentgen examination six months after the first (Fig. 253) shows the deformity of the duodenum practically unchanged. In this case surgery was advised on account of the partial success of medical treatment, but the patient would not consider it.

Synopsis of Medical Treatment.—*Rest in Bed.*—This is needed for one to four weeks in the more severe cases, particularly the gastric ulcers, and all cases where there is much pain or such complications as obstruction, hemorrhage, or malnutrition. I always prefer a week in bed for all ulcer cases, but it is not absolutely necessary in the milder type of case, particularly the duodenal ulcer in vigorous men without complications. I have usually obtained excellent results in these cases by the use of diet and drugs without the necessity of taking the patient entirely away from his business and putting him to bed.

Local Applications.—A Preisnitz compress changed every twelve hours is frequently useful in relaxing spasm and making the patient comfortable. If there is pain, a flaxseed poultice may be used.

Diet is subject to more hobbies than any other part of the treatment; we must remember that radically different diet schemes in the hands of experts show very small variation in the percentage of cures. We have tried to make no restrictions that are not needed.

In the more serious cases we give two or three days of exclusive rectal feeding at the start, in order to rest and quiet the stomach, lower secretion, lessen peristalsis, relax spasm, and get the stomach entirely empty. We use 8 or 10 ounces of 10 per cent. glucose solution in water, to which 1 ounce of 50 per cent. alcohol may be added (Smithies). If necessary, 10 drops of tincture of opium may be put in every second or third enema. This is given by the drop method three or four times a day. Rectal feeding gives more water than food, but about 500 calories are absorbed, and the glucose prevents starvation acidosis.

When little or no food is given, chewing paraffin wax a half

dozen times a day keeps the mouth clean, stimulates the flow of alkaline saliva, and lessens hunger contractions and thirst.

We use chiefly carbohydrate foods at first because they cause little acid secretion and peristalsis. The pylorus is relaxed and opens easily. This keeps the stomach empty and the acids low. We use chiefly cereal gruels and cream soups or powdered crackers and milk. Milk is better given in cereal gruels or with egg, or predigested, or mixed with cream or crackers, on account of the tendency of plain milk to form hard curds in a sour stomach. The diet is a much modified Lenhartz. The following is the usual scheme:

First Week.—Strained cereal gruels or strained cream soups made from vegetables, or milk and cream equal parts, or powdered crackers and milk are given in hourly feedings, beginning with 1 ounce and increasing up to 3 or 4 ounces. If desired, the Lenhartz feeding may be given, namely, 8 ounces of milk and 2 eggs beaten up and divided into twelve feedings, one every hour. Each day add 1 more egg and 3 ounces of milk, until 6 eggs and 1 quart of milk are taken. On the third day add ½ ounce of sugar or milk-sugar to the beaten eggs, gradually increasing to 1 ounce of sugar.

Second Week.—One-half the eggs (3) may be soft cooked; three feedings of 1 ounce each of boiled rice or other well-cooked cereal (not oatmeal) served with sugar and cream. One-half to 1 ounce of butter per day. Gruels, cream soups, egg and milk, one or two pieces of very dry toast well chewed, or milk-toast, macaroni, Mellen's Food, or malted milk, custard, cream-toast, ice-cream. Six meals a day.

Third Week.—Add 1 or 2 ounces of scraped beef or tender chicken, chop or steak daily, also simple cornstarch, custard, and rice puddings.

Fourth Week.—Add well-cooked mashed vegetables; vegetable purées; apple sauce or stewed pear; finely chopped spinach.

After Fourth Week.—The foods to take are: milk; butter and cream soups; cocoa; soft cooked eggs in any form, two at a time, may be eaten as often as twice a day. Fish must be fresh and always broiled, baked, or boiled—never fried. Fresh beef,

lamb, mutton, or chicken may be eaten in moderate amounts once a day. The meats should be as tender as possible, always roasted or broiled, not cooked too much, cut fine on the plate, and well chewed before swallowing. Toast, crackers, breads, rolls, and plain cake when not too fresh are allowable at all of the meals, and plain fresh butter in large quantities. The cereals—cornmeal, hominy, cream of wheat, farina, rice—must be well cooked. The best vegetables are rice, mashed potatoes (white) or baked sweet potatoes, asparagus tips, young peas, chopped spinach. Any dessert made of cereals, butter, milk or cream can be used (custard, simple puddings), also simple jellies. Fruits—apple, pear, peach, prunes, apricots—should be baked or stewed. The best drink at meals is plain cool water or hot malted milk or cocoa.

General Directions.—Eat slowly, chew well; no very hot or very cold food; no worry at meals. Never eat until overfilled. Take a little lunch between meals, for example, a glass of malted milk or cocoa with one or two crackers. No raw fruits or vegetables for six months.

For the *ambulatory cases* we use the following scheme:

The general plan of the diet is three moderate-sized meals at regular intervals in the day, and supplemental meals of malted milk or milk reinforced with cream, cocoa, and crackers between meals and before retiring. All of the solid foods should be tender, cut very fine on the plate, and thoroughly masticated before swallowing. Altogether, foods should be taken at least five times in a day in one or the other of the two types of meals. No meat broths, condiments, spices, coffee, "soda water," alcohol, coarse or salted food, very hot or very cold foods.

The foods permitted for the main meals are cream soups. Eggs in any form about four in a day. Cereals (well cooked) with cream in the morning. Macaroni, vegetables, any that are well cooked and mashed, such as potato, squash, rice, finely chopped spinach (no green vegetables, fruits, berries or nuts). Desserts, any made of milk, eggs and cereals (custards, simple puddings), and jellies, cream cheese, cooked fruit—apple, pear, peach, prunes. The best drink at the meals is water, hot cocoa,

or malted milk. Butter, cream, milk, eggs, cereals, and bread remain the main foods of the diet. Olive oil should be taken after the main meals, a dessert or tablespoon.

After the first or second week fresh beef, lamb, or chicken roasted or broiled, or fresh fish broiled, baked, or boiled may be taken once a day.

The Stomach-tube.—This may be needed for the first few days *in cases with marked spastic obstruction* to help keep the stomach empty. There need be no hesitation in using it for this purpose in ulcer cases. I have never seen it do harm in a mechanical way.

In general, with atropin and a moderate use of alkalies, together with a bland diet and plenty of fats in small repeated feeding, the tube is not often needed.

If distress occurs in spite of very careful treatment, it is always fair to use the tube to see why (gastric stasis, overacid contents, etc.).

The Duodenal Tube.—I have given the duodenal tube a thorough trial in chronic ulcer, and no longer use it. It gives good results, I can vouch from my own experience, but I get just as good or better results by the treatment already outlined, with less discomfort to the patient. The duodenal tube has helped me more in cases of persistent vomiting in nervous women than in any other class of case.

Drugs.—The most useful are the alkalies and atropin.

Alkalies.—We give them on the theory that distress or pain is due partly to increased peristalsis and muscular spasm, and thus to delay in emptying the stomach and partly to irritation of the ulcer by free acid. Our object is to neutralize free acid and check spasm and help the stomach to empty. Nothing is more definite in medicine than the relief of hunger-pain in ulcer by alkalies. It probably is not necessary to neutralize every trace of free acid to get these good results. The use of excessive amounts of bicarbonate of soda may even irritate the stomach. Soda has been much discussed, and experts get excellent results in treating ulcer using either little or much, which shows that the amount given is not vital. I believe the middle

course is the best. Five to 10 grains of calcined magnesia is often better than bicarbonate of soda, whose neutralizing power is low, about one-quarter that of magnesia, and forms troublesome gas (CO_2). Many cases need no alkalies if the diet is properly regulated.

I believe that the scheme that Sippy has so much emphasized of giving small amounts of bicarbonate of soda and oxid of magnesia between meals at first is a good one. We may use milk of magnesia, 2 to 4 teaspoons, instead, and we can alternate the magnesia with a mixture of subcarbonate of bismuth, 10 grains, and sodium bicarbonate, 20 grains, if the bowels become loose. Some objection has been raised to this alkaline treatment on the ground of irritation of the stomach and hydrorrhea, but I find that this rarely occurs, and when it does appear, soon passes off.

The common fault of the family doctor in these cases is to use too little alkali, and to *wait till pain appears and then relieve it.* This is a serious mistake. It is very slow work healing an ulcer in this way. We should aim to *prevent the appearance* of these painful periods of spasm and peristalsis and acidity by frequent small feedings of bland food and by the use of alkalies *in advance of the customary periods of hunger-pain.* Patients are often "afraid soda will hurt the lining of the stomach," and need to be encouraged. A few patients with ulcer have habitually low gastric secretion; here alkalies are not needed.

Atropin, $\frac{1}{150}$ grain, two or three times a day, is very useful in relaxing spasm and checking secretion, and the bedtime dose is one of the best means of getting the patient comfortably through the longest interval between meals. I see no advantage in giving it subcutaneously except in cases of vomiting or hemorrhage, the physiologic result is so readily obtained by mouth.

Hydrogen peroxid promised well as a powerful means of checking gastric secretion, but it has proved too irritating, even when well diluted, and I have given up using it.

Bismuth is much used, and probably has some effect in protecting an ulcer. It is not important. In a series of test cases, giving every other case bismuth, no difference in the result was

noted. It is valuable as a corrective if much magnesia has to be used.

Tobacco.—If the patient is a smoker, it is very desirable to greatly reduce or omit tobacco.

Hemorrhage.—We want a quiet contracted stomach, to let the open blood-vessel retract and close the folds of mucous membrane and favor clotting. We want low or moderate blood-pressure. These indications are best met by a subcutaneous injection of $\frac{1}{4}$ grain of morphin with $\frac{1}{30}$ grain of strychnin, to keep the stomach contracted. Absolute rest in bed, no food by mouth for a few days, no vomiting, no unnecessary handling, no lavage, no rapid introduction of fluids, no stimulation unless the blood-pressure falls very low. We keep up the body warmth, and furnish some fluid by rectum or intravenously if absolutely necessary. A large enema of normal salt solution is usually undesirable. It fills the vessels, raises pressure, thins the blood, and lessens its coagulability.

In hemorrhage cases we always begin with rectal feeding alone, but have no hesitation in giving food by mouth after two or three days.

Lavage is dangerous and very undesirable, as a rule, on account of gagging, increase of blood-pressure, and peristalsis; cases are reported where the tube has helped by emptying a full bleeding stomach just as we empty a full uterus, but I have not seen one.

One of our first thoughts as we stand beside a bleeding ulcer patient is that we *must* stop the bleeding at once. Our second thought is that the various vasoconstrictors and coagulants are not very reliable. There are plenty of platelets and coagulating ferments in the blood, and little or nothing is gained by adding more.

We usually give $\frac{1}{2}$ to 1 teaspoon of a 1 : 1000 adrenalin solution in $\frac{1}{2}$ ounce of ice-water by mouth for local action in the stomach if the bleeding continues. It should not be used intravenously, as it promptly raises the blood-pressure.

With regard to coagulants, we know there is a natural steady increase in coagulability in hemorrhage. There is no lack of

any of the coagulation elements and the coagulation time is normal or less. Coagulants may be useful in small continued oozing or in preventing a second large hemorrhage, but the doses need to be given frequently, as their action is very brief.

There are many difficulties with blood-serums. The danger of severe anaphylaxis; they are not powerful coagulants; they rapidly deteriorate and are of little use after a week or two. It takes some hours to prepare serums freshly, and about a day passes after their subcutaneous use before they effect clotting. Fresh rabbit serum is the most useful, it is active and easy to get, and causes less anaphylaxis. It is given subcutaneously 10 to 50 c.c. every six to twelve hours for one to two days. The serum of stock diphtheria antitoxin has almost no value.

Coagulose made from horse-serum keeps for a long time and is active; 10 grains added to 8 c.c. of cold sterile water represents 10 c.c. of serum. It can be given subcutaneously, or into the muscle, or into the vein. It cannot be sterilized by heat. I always feel a little doubtful about the usual preparation, that is little called for and has been on the druggist's shelf for a long time.

Coagulen, a powder prepared from blood-platelets, is easily soluble in water and may be given by mouth for local action in the stomach in 1- to 3-ounce doses of a 10 per cent. solution in water. It is little used at present.

Calcium salts are hopeless in acute hemorrhage. They must be given in very large doses for many days.

The value of transfusion *in acute first hemorrhage* is under discussion, with the odds in favor of transfusion. This is more useful as a method of general help to a weak, anemic patient than as a method of stopping acute bleeding from an ulcer.

Surgery.—We always want medical treatment for *acute* hemorrhages from the stomach. It was only a few years ago when not a single patient in the two largest hospitals in Boston had survived an operation for acute hemorrhage from the stomach.

Nearly all acute ulcer hemorrhages stop under medical treatment and, as Moynihan says, not over 3 per cent. of freely bleeding ulcers could be saved by surgery at the time.

Recurrent hemorrhages, or oozing of blood which does not yield promptly to medical treatment, demand surgery. If such patients could be thoroughly examined, definite reasons for surgery could usually be found—large gastric ulcers, obstruction, etc. With the present intensive study which is made of chronic ulcer cases, very serious or fatal hemorrhages are becoming more and more rare. The serious cases are weeded out and dealt with more thoroughly and much earlier, and the risk of serious hemorrhage is greatly reduced. I have seen only one fatal hemorrhage from ulcer in the last seven years, and in this case there had been repeated warnings which, if attended to, I believe would entirely have prevented the fatal outcome. The persistence of occult blood for several weeks in spite of careful medical treatment makes one suspect cancer and justifies exploratory operation as a rule.

CLINIC OF DR. JAMES P. O'HARE

PETER BENT BRIGHAM HOSPITAL

CHRONIC NEPHRITIS WITH EDEMA

Report of a Case Terminating Fatally; Many Interesting Features, and Full Report of Autopsy.

IN this clinic I am going to discuss a case of chronic nephritis which has recently come to autopsy. The patient, a girl of ten, entered the Peter Bent Brigham Hospital August 31, 1917, and remained here continuously until her death on October 9, 1918—a period of more than thirteen months. During this time she has been under constant observation and study, as a result of which much useful data has been accumulated. Some of this can only be touched on here.

The past history of this little girl was unimportant except for an attack of "liver trouble" three years before entrance. This lasted for six months, was accompanied by jaundice, and kept her confined to bed almost all of that time. Now, this trouble may be of some significance as a possible cause of the onset of her nephritis, especialy since she gives an indefinite history of nocturia for one year. As you know, nocturia means awakening out of sleep to pass a goodly quantity of urine. It does not include voiding at night by a patient who cannot sleep. Accepting this definition then, nocturia is of considerable significance in determining the onset of any nephritis. Necessarily the nephritis antedates the appearance of this symptom. In our patient I have some doubt about the accuracy of the history of nocturia because during the patient's hospital career she rarely had this symptom.

The more probable etiologic factor in her was a rather mild

attack of diphtheria three months before coming to us. During this illness she was at the Boston City Hospital, and was discharged well. Incidentally, her family blames the antitoxin for the nephritis, whereas it was, of course, the diphtheria toxin.

Two weeks after leaving the city hospital her eyes became puffy. (Note that time—two weeks. The first symptom observed in most of our cases of acute nephritis is swelling of the eyes or feet, coming on ten days to two weeks after the patient is apparently well of the causative infection. The latter has usually been a throat or upper respiratory infection.) About one week later our patient noticed edema of the legs and—shortly—abdominal and facial swelling. No hematuria, headache, or drowsiness was noticed. She did, however, become nauseated and vomited a few times. She was compelled to go to bed, and two weeks before entrance 4 quarts of fluid were removed from her abdomen. She came to us for "swelling."

At entrance she showed general edema of the subcutaneous tissues reaching on the trunk as high as the manubrium sterni. The face was puffy, the legs were swollen, and there was considerable fluid in both pleural cavities and in the abdomen. The latter was tapped for relief almost at once.

Apart from the edema, physical examination showed but little. There was the characteristic pale, pasty expression to go with a somewhat lowered hemoglobin and an edematous face. Several teeth were necrotic. The tonsils were not at all enlarged. Bear in mind, however, that the small buried tonsils may be very much more dangerous than the large ones with plugged crypts.

The heart was of approximately normal size; the apex impulse being felt 9 cm. from midsternum, corresponding to the left border of dulness. The right border was percussed 3.5 cm. from midsternum. There was a soft systolic murmur at the apex. The radial arteries were apparently normal. The blood-pressure was 165 systolic, 95 diastolic. The urine showed a large trace of albumin, many granular casts, and a few red and white blood-cells.

Ophthalmoscopic examination made shortly after entrance showed no abnormality.

The picture, therefore, at this time was that of a subacute nephritis following diphtheria.

The patient's course in the hospital was essentially one of storing up fluid until she had to be tapped, and then refilling slowly again. As time went on the fluid accumulated less in her subcutaneous tissues and more in her abdominal cavity. In all, her abdomen had to be tapped fourteen times in fourteen months. There was nothing else in the course of her disease that was particularly unusual. She had an occasional headache and was easily nauseated and vomited. These symptoms, however, were usually prominent only when her abdomen was much distended with fluid. She was at all times rather weak. On the whole, it may be said that her hospital life was characterized chiefly by a series of abdominal taps.

In the last quarter of her stay with us her renal function tests had been showing a progressive decline. Two weeks before her death she had a slight but rising temperature. With this there developed an area on her cheek suggesting, very slightly, erysipelas. (I rather doubt that it was this disease.) At once her face became terribly swollen and the edema elsewhere increased rapidly. She voided less and less and was practically anuric for several days before death. In spite of this extreme oliguria the patient had but few symptoms. The urine picture changed to one indicating great degenerative changes in the kidney. The fields of the microscope were filled with casts of all kinds: hyaline, granular, and cellular. There was but little blood. The blood urea nitrogen had climbed rapidly to 90 mgm. and the phthalein excretion would undoubtedly have been zero if we could have made the test. (Output of urine was too low.) Toxic symptoms—severe headache, drowsiness, restlessness, urinous breath, terrible nausea, vomiting, and twitching—then appeared. Her heart became very rapid and irregular; she developed Cheyne-Stokes' respiration and died, retaining consciousness to the end. There was no evidence of any acidotic breathing and there was no acidosis. There were no convulsions.

Now, the outstanding interest in this case centers, of course, in the edema and the salt-and-water metabolism. Let us postpone this, however, until we have considered some of the other features.

The results of routine urinalysis were interesting. The specific gravity averaged about 1019 all along. This is pretty high concentration for such badly damaged kidneys as the pathologist found. Within ten days of her death she was able to concentrate to 1025 for a twenty-four-hour period. There was usually a slight trace to a trace of albumin, numerous hyaline, less numerous granular, and occasionally a few cellular casts. Red cells were numerous at first and gradually tended to disappear. White cells, which were also numerous, decreased more slowly. As has been stated before, during the last two weeks of life the kidneys were undergoing rapid degeneration. The urine, as a result, was loaded with casts of all kinds: hyaline, and the more significant coarse brown granular, and cellular casts. Curiously enough, there was but little blood.

Table I is a summary of the other features, and is taken from charts which are too cumbersome for this exercise. As you see, the patient's thirteen hospital months have been roughly divided into four equal parts. This is as good a division as any, inasmuch as there were no special episodes to make natural divisions. Let us consider some of the different items.

Take the blood-pressure first. On the whole, the diastolic paralleled closely the systolic, so that I shall consider only the latter. This pressure fluctuated moderately from day to day, with changes only indirectly connected with the disease. For example, there was a rise when the patient was up and about and a fall when she was put back to bed. There was also a slight rise when the patient's abdomen was much distended. Apart from such changes, however, the systolic curve is what you would expect. At entrance the pressure was moderately elevated, averaging about 160 during the first ten days. It then fell to around 135, where it remained for the greater part of a year. Now, remember that while a systolic pressure of 135 is

TABLE I

		First quarter.	Second quarter.	Third quarter.	Fourth quarter.	Maximum variations.
Blood-pressure, systolic	Maximum,	165	145	150	202	110–202
	Minimum,	111	120	125	110	
	Average,	135	135	135	150	
Blood-pressure, diastolic	Maximum,	100	110	115	130	70–115
	Minimum,	72	70	80	80	
	Average,	90	90	90	105	
Phthalein	Maximum,	43	55	38	34	9–55
	Minimum,	28	25	23	9	
	Average,	37	36	30	20	
Blood-urea, nitrogen[1]	Maximum,	77	27	41	122	11–122
	Minimum,	22	14	15	11	
	Average,	26	18	20	17	
Blood chlorid[2]	Maximum,	5.5	5.8	5.8	6.00	4.75–6.00
	Minimum,	4.8	5.6	4.75	4.85	
	Average,	5.2	5.35	5.25	5.50	
Urine volume	Maximum,	1375	650	1025	1400	0–1400
	Minimum,	100	150	150	0	
	Average,	400	300	300	750	
Urine chlorid[3]	Maximum,	0.54	1.1	0.56	2.88	0–2.88
	Minimum,	0.04	0	0	0	
	Average,	0.16	0.8	0.06	0.8	
Urine nitrogen[3]	Maximum,	12	4	10	8.8	0–12
	Minimum,	0.4	1.2	1	0	
	Average,	3	2	2	4	

[1] Mgm. per 100 c.c. [2] Grams per liter, whole blood.
[3] Grams per twenty-four hours.

normal for an adult, it is not so for a girl of ten. Her normal should be around 100.

During the last quarter there was a steady rise in pressure, reaching a maximum of 202 at about the onset of her final stage. This, of course, is very high, and is equivalent, in an adult, to something like 260. Such a pressure is unusual in a true diffuse chronic nephritic. It is more like what we see in the straight vascular hypertensive case. Curiously enough her blood-pressure fell in the terminal stage to 135 and then rose again to 175 two days before her death. This drop before death is not very

unusual and is probably due to a relaxation of the whole vascular system.

In general, therefore, it may be said that the blood-pressure was only moderately elevated during the greater part of the disease, was higher in the active stage at entrance, and extremely high a short time before death.

If you look at the excretion of phenolsulphonephthalein you will see that, on the whole, our patient did pretty well. During the first three-fourths of her hospital life the average excretion was 30 to 35 per cent. instead of the normal 60. Now this is not at all bad, and a glance at the maximum and minimum figures will show that at times the kidneys handled this dyestuff almost as well as normally. At one time the patient excreted 55 per cent. in two hours and ten minutes. Bear in mind, however, that a pathologic kidney under no strain may show a normal phthalein excretion, but when the strain is applied the function may be much depressed. We have recently had a patient in the hospital who had a phthalein excretion of 45 per cent. and a normal blood urea and blood chlorid. He was operated on for his hematuria and a large polycystic kidney removed. There was not one bit of functionating renal tissue left. The other kidney was normal and was doing all the work. If you remove one kidney in a dog, the other will function normally under average conditions. Even in a diseased kidney the extent of the damage is not accurately gauged by the usual functional tests performed under conditions of comparative rest. In such a kidney the remaining good tubules and glomeruli hypertrophy and take on the burden of the damaged or destroyed ones. When these are pushed beyond their capacity, however, they give way, and the function, which has been good, becomes distinctly poor. The kidneys of our patient were doing a minimum of work and getting a maximum of rest on a low protein and low salt diet, etc.

In the last quarter there was a gradual but well-marked fall in the phthalein excretion—no doubt to zero if we could have performed the test at the end. Incidentally, it was during this period that the kidneys were put to the greatest strain by a

long period of high protein diets. I do not believe, however, that this could have had much influence on the phthalein excretion or on the outcome, since her kidneys, during this period of high feeding, did better work than at any other time in the disease. I refer to the excretion of water, salt, and nitrogen.

The blood urea nitrogen curve is of significance for three reasons: In the first place it was normal or nearly normal during the greater part of the disease. It shows conclusively that this substance may be normal when the function of the kidney in other respects is almost nil. For five weeks during which the phthalein excretion averaged 14 or 15 per cent. and even as low as 9 per cent., the blood urea nitrogen never went over 16 mgm. (15 mgm. per 100 c.c. is assumed to be the high limit of normality under average conditions). This points to the fact that the blood urea nitrogen is in itself not a sufficient test of renal function. When combined with the phthalein excretion we think it is quite so, even better than the more complicated Ambard or urea index.

In the second place this urea curve shows that this substance is dependent on the intake of nitrogen in the diet plus the breakdown of tissue protein. In our patient there were but three periods during which the blood urea nitrogen was much elevated. The first two correspond accurately to the taking of the high protein low fat diet of Epstein and the last, at the end of life, when there was a tremendous breakdown of body tissue. As a result of the latter there was a rapid rise in two to three weeks from normal to 122 mgm. per 100 c.c., *i. e.*, about ten times the normal.

A third thought is brought out in the comparison between the blood urea nitrogen and the phthalein curves. We are in the habit of assuming that as one falls the other rises. This is true in experimental acute nephritis in animals, and it seems to be true in many patients for a short time after they enter the hospital. With a low protein diet and rest in bed they begin to get rid of the excess nitrogen that they have been accumulating before entering the hospital. With this comes a relief of pressure on the kidneys and the function improves. In such a

case the phthalein will rise as the blood urea falls. There is really no relation between the two; at least in our case. The two were quite dissociated. With the fall in the phthalein from near normal to zero the urea in the blood remained at or near normal. Furthermore, in the episode of Epstein high-protein feeding, when the blood urea nitrogen rose the phthalein actually rose with it.

The urinary nitrogen curve is of no great significance, and there is but little excuse for doing this rather tiresome and monotonous grind. In this case the intake of nitrogen was low, the output was about normal. When the intake was raised the kidneys tried to follow. In this they were not very successful. On an intake of nitrogen of 3 to 4 gm. during the time when the patient was on a low protein diet, the kidneys excreted 2 to 3 gm. If you remember that 1 gm. or even more of nitrogen may be lost in the feces, you will see that the excretion of nitrogen by the kidneys was very good. When, however, the higher protein diets were fed the kidneys could not do as well. On an intake of 10 to 12 gm. they excreted only about 6 gm. It may be of interest to note that during the two Epstein diet episodes these kidneys were able to excrete as high as 10 to 12 gm. of nitrogen in twenty-four hours on occasions.

Now to consider the most interesting part: the salt-and-water metabolism. Let us take the blood chlorid. In most cases we feel that the determination of this substance reveals but little to us. This is due to the fact that the laws governing the concentration of salt in the blood are not at all established. Furthermore, it is known that certain drugs may change the concentration of this substance by displacement of it out of the blood-stream, presumably into the tissues. Outside the body, too, if one is doing the chlorids of the plasma, there may be an absorption of salt by the corpuscles if the blood is shaken too vigorously with air. The determination of the chlorids of the whole blood, rather than those of the plasma, obviates this last difficulty. In fact, it has been necessary for us to do this because we were unable to obtain blood charcoal.

In our case the blood chlorid was of considerable interest

in one or two ways. Its concentration was always somewhat elevated. The threshold of the kidneys for salt was undoubtedly raised. You know, of course, that the so-called "useful" substances in the blood, those like salt and glucose, which the body can keep on using, are supposed to have a threshold. When the concentration of these substances in the blood reaches a certain level they are no longer excreted in the urine. The "useless" substances, like urea, have no threshold, but are removed from the blood-stream as fast and as completely as the kidney can do so. Does it not seem extremely significant in our case that the chlorid was highest in the blood during the only time when the excretion of salt and water by the kidneys was good? This, too, was when the patient was taking a mixed diet high in protein, but low in salt. It would seem to point to the possible conclusion that the tissues were more at fault than the kidneys. It therefore seems fair to conclude that a combination of water and chlorid, perhaps with something else, occurred in the tissues, and that the feeding of the higher protein diets loosed this combination. Additional weight is given to this hypothesis by another experiment. While our patient was excreting but little salt and water we fed her considerable amounts of sugar in the form of candy. At times it seemed as though there resulted a short-lived increase in the excretion of salt and water. We are rather conservative about such interpretations, however, since we have found, all too often, changes in the excretion of different substances without any known cause. On occasions we have noted well-marked changes in the excretion of water, salt, and nitrogen on days when we had planned to carry out a certain procedure, but were prevented from so doing. You see how easy it is to misinterpret moderate changes.

Take next the urine volume. During the greater part of the hospital life she excreted a very small amount. On an average intake of 1000 c.c., the output equalled only 300 to 400 c.c. The difference was largely stored in her abdomen. There were three or four exceptional periods, however, during which the patient excreted a considerable amount of urine, in fact, had a diuresis. These periods were coincident with the giving of a diet high in

protein, either the Epstein diet or our own standard nephritic salt-poor diet. This is worth remembering, since we do not often think of giving our nephritics a high protein diet. Curiously enough, the Epstein diet increased the outflow of water, but did not affect the chlorid excretion. On the other hand, our mixed standard nephritic diet with a lower protein content produced a greater excretion of both salt and water. During less than a month of this diet, from July 26th to August 16th, the patient excreted 29 gm. of chlorid. This is only 9 gm. less than the entire chlorid excretion of the previous eleven months in the hospital. We had tried renal stimulants of all kinds, calcium lactate, purges, etc. but none of these had any appreciable effect on the water metabolism.

The chlorid output was as interesting as the water. We ordinarily think of these two as going hand in hand. But, as you have seen, it was not so in this case. The amount of salt excreted was extraordinarily small in comparison to the intake. The latter averaged about 2 gm. per day. In the table you will see that during the first nine months our patient excreted on the average not more than 0.1 gm. per day. In fact, there were many days (once five days in succession) when not a bit of chlorid could be obtained in the urine.

Where did this salt and water go? A glance at Table II will show you. This is a summary of the tappings, the volume of fluid withdrawn, the chlorid content of that fluid, the total urine chlorids excreted between tappings, and the approximate intake during these periods.

In ten months during which comparisons were made the patient took by mouth about 517 gm. of chlorid.[1] In that same period 418 gm. were recovered from the abdominal cavity, while only 98 gm. were excreted in the urine.

Just look at the column giving the figures for the volume of fluid removed from the abdomen. The patient was tapped in all fourteen times in as many months. If we include the fluid obtained at autopsy there was removed 96,500 c.c. This is

[1] See Table II, December 10th to September 27th.

TABLE II

Date of tappings	C.c. removed.	Cl. in fluid, gm.	Cl. intake, gm.	Cl. in urine, gm.
August 16th[1]	4,000			
September 1st	5,200			
October 1st	4,500		58	7.29
October 29th	5,700		56	6.54
November 13th	6,175		30	2.09
December 10th	4,800	32	56	3.66
January 10th	5,900	40	53	2.0
February 4th	6,200	41	48	1.7
March 7th	7,200	47	62	5.0
April 8th	6,785	43	64	5.0
May 2d	7,230	48	48	1.5
June 20th	10,000	69	60	3.0
July 17th	9,050	64	54	11.0
September 27th	8,260	54	72	65.0
Postmortem	5,500	24		
Total	96,500	462	661	113.78

[1] Before entrance to hospital; amount only approximate.

equal to well over 200 pounds, or three times the body weight of the child.

It may be of interest to know that the ascitic fluid in this case was always of the opalescent gray type, resembling, in its appearance, of course, chylous ascitic fluid. This sort of fluid has been typical of all of the nephritics we have seen in the last two years. In connection with the removal of the fluid in the abdomen the basal metabolism is of interest, too. It was found to be very low (—35) just before tapping. We thought that the removal of the abdominal fluid might raise this to nearly normal, on the assumption that the patient's true body weight —one of the figures used in the calculation—was more nearly correct after the removal of the fluid. This did not prove to be correct. The metabolism determined the morning after the fluid had been removed showed practically no change (—35 to —33).

The creatinin in the blood is supposed to be of assistance in determining the prognosis of a given case. This is based on the theory that this substance is handled with the greatest ease by the kidney. When, therefore, this begins to accumulate in the

blood it indicates that the kidney is badly affected. In our case it did not help us much. The blood creatinin was only 2.35 mgm. per 100 c.c. within twelve days of her death. With the rapid breakdown in tissue protein before death it rose rapidly, as one would expect, to 14 mgm. per 100 c.c.

Before speaking about the postmortem examination I should like to say a word about the eye-grounds in this case. These were examined repeatedly, and they seemed to be perfectly normal in every respect until the end. About ten days before she died she had blurring of her eyes, and examination showed moderate blurring of the optic disk in both eyes and a few spots of exudate in one eye.

We were indeed fortunate in getting an autopsy to complete this case. This, too, showed many interesting features. In the peritoneal cavity there were 5½ liters of fluid. The omentum was much thickened, as was the peritoneum everywhere. This was brought out strikingly in the comparison between the serous surfaces covering the two sides of the diaphragm. The peritoneum was about three times as thick as the pleura opposite it. This is of interest because, you will remember, the fluid kept accumulating more and more in the peritoneal cavity. The pleural cavity was only occasionally affected. Whether this thickening of the peritoneum was responsible in great part for the deviation of most of the stored water and salt into this cavity or whether the thickening was secondary to the accumulation of fluid here is hard to determine. In this regard, the fact that the whole peritoneum seemed uniformly thickened and that those parts that were presumably most in contact with this fluid were no more thickened than the other may be of some importance.

The pericardial cavity contained a slight excess of fluid.

The heart was uniformly hypertrophied, and the chambers, especially the left, somewhat dilated. The valves were normal and the myocardium showed nothing except that the muscle-fibers were larger than one would expect for the age of our patient.

The aorta throughout its whole length together with its

main branches showed numerous yellow plaques of arteriosclerosis. The renal and splenic arteries were especially bad.

The spleen and liver showed chronic passive congestion. All of the other organs with the exception of the kidneys were normal.

These were very small, one weighing 95 gm. and the other only 65 gm. They were grayish-red in color, with smooth surface. The cortex was considerably thinned and there seemed to be considerable fat and fibrous tissue. On microscopic examination there was extensive fibrosis of the cortex and medulla. Many of the glomeruli were entirely replaced by fibrous tissue. The rest showed "all stages of intracapillary and capsular proliferation." There were none that were normal. The common lesion was a hyaline thrombus in the capillary with proliferation of the capsular epithelium. The tubules were tremendously atrophied. The arteries showed extensive lesions with many fresh thrombi and considerable sclerosis.

Summary.—In this clinic I have presented to you a case of chronic nephritis with edema, which came to us originally as a subacute nephritis following diphtheria. The patient showed a moderately elevated blood-pressure and at the very end a very high one. The renal function tests exhibited only moderate disturbances until near death, when the function became extremely poor. The chief interest in the case lies in the inability of our patient to handle water and salt. These two substances after a while were stored almost entirely in the abdominal cavity. Numerous tappings had to be performed for relief. The only periods during which our patient handled water and chlorid well were coincident with the feeding of a diet high in protein. No success was obtained in getting rid of a fluid during the periods of low protein salt-poor diets or, in fact, in any other way except tapping.

Postmortem showed a marked glomerular nephritis, hypertrophied heart, and extensive arteriosclerosis.

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SOME LESSONS OF THE WAR IN THE FIELD OF CARDIAC DISEASE

THE experiences of medical men during the war have thrown much light on the subject of cardiac disease, and the knowledge gained is of value not only in the handling of soldiers but also in the care of civilian patients. It seems worth while, therefore, to call attention to some of the points which have been learned and to some of the problems that have arisen, so that we may carry back into our civil practice as much as possible of what we have learned during our service in the army. The patients to be discussed at this clinic are all discharged soldiers. The first illustrates the functional efficiency of certain well-marked cases of valvular heart disease. The second is to call attention to a stage of valvular heart disease rarely seen in civil practice, and to show the difficulty of diganosis in early mitral insufficiency. The third is to show a condition bearing superficial resemblance to heart disease and now generally known as the "effort syndrome" or the "irritable heart of soldiers." It is a type of case which became particularly prominent during the war, but which exists potentially, and often unrecognized, in civil life. Certain types of this condition are the direct product of the stress and strain of war, but in our army most cases have shown symptoms which antedated the mobilization by many years.

Owing to the careful and for the most part skilful work of the Cardiovascular Boards which examined the recruits only a very few men with valvular heart disease were accepted for service. Those who slipped by were, for the most part, men

without symptoms, and in whom the signs were obscure and difficult to recognize. It is indeed remarkable that, considering the haste and the difficult circumstances under which the examinations were carried on, so small a number of mistakes were made. Nevertheless a few men with heart disease were accepted. It is, however, probable that most of these were passed in the early weeks of the recruiting, before the examinations reached the high state of efficiency to which they finally attained.

CASE I.—The young man whom I wish to present to you first was invalided home from France in the summer of 1918, and was sent to the General Hospital at which I was on duty. He presented himself to me one day and reminded me that four years ago he had been a patient in my ward at the Peter Bent Brigham Hospital in Boston. He told me that he had been in the hospital on account of heart disease. Briefly his history in the army had been as follows: He had volunteered in the fall of 1917, had been hastily examined, accepted, and sent to France. There he had undergone strenuous training until he was sent to the front, where he "carried on," doing full duty without any difficulty for five months. During this time he hiked with full equipment. When asked as to the strain under which his heart gave out, he said: "My heart didn't give out at all. I got gassed. Then I was sent to a hospital, where they found a murmur and sent me home." His general condition bore out his story, for he was in excellent shape, and was able to take the severest of the graduated exercises without any difficulty. Let us now consider for a moment his past history. I turn to the record made in September, 1914, at the Peter Brigham Hospital, when he was fourteen years of age. The diagnosis at that time was "rheumatic fever; chronic cardiac valvular disease; aortic regurgitation; mitral regurgitation, and delayed conduction time." The previous history was as follows: In 1909 he was in bed for three months with an attack of rheumatic fever in which nearly all of his joints were involved. The following year he had a recurrence of the rheumatism which again involved all his joints and kept him in bed for four months. The next year he had a third attack of rheumatic fever, after

which he noticed that he became short of breath rather easily. In the summer of 1913 he had a fourth attack, in which, however, only one ankle was affected. After this he was up and about and was not bothered by any marked shortness of breath. The illness for which he entered the hospital began early in September, 1914, with a sore throat. Following this his ankles became swollen and stiff and it hurt him to walk. He did not feel feverish until the day before admission and he kept up and about until then. On the day before admission he began to have pain in the left side of his chest and some dyspnea. During the night he was unable to sleep lying flat. On physical examination he was found to be moderately dyspneic and very slightly cyanotic. The heart's action was regular and moderately rapid. The apex impulse was felt in the fifth interspace 12 cm. to the left of the midsternal line, and the cardiac dulness extended from 3 cm. to the right of the midsternal line to 13 cm. to the left in the fifth space. At the apex the first sound was followed by a long blowing systolic murmur which was transmitted toward the axilla. In the aortic area no second sound was audible, but there was a rough systolic murmur transmitted into the vessels of the neck, and a diastolic murmur which was loudest in the third interspace to the left of the sternum. The lungs were clear and the abdomen negative. The liver was not enlarged. There was a moderate amount of edema over both tibiæ. The electrocardiograms showed a prolonged conduction time. The blood-pressure was 120 systolic and 46 diastolic. During the first part of his stay in the hospital he had a moderate degree of fever, but his temperature soon reached normal, and with rest in bed and a little digitalis his heart became fully compensated. He was in the hospital for over two months, and on the last note before his discharge the border of the heart was made out to be 12 cm. to the left of the midsternal line in the fifth space. The electrocardiograms showed that the conduction time had become normal in length. At the time of his discharge he had little dyspnea or rise of pulse-rate when walking about the ward. He had made an excellent recovery, and left the hospital in good general condition.

Here, then, was a boy with a history of multiple attacks of acute rheumatic fever which had left him with a heart markedly enlarged and with perfectly evident signs of aortic and mitral insufficiency. He was hardly the material out of which one would expect to make a good soldier, and it is, of course, rather extraordinary that he was able to pass the physical examination of the army. On his return to Boston, after being discharged from the army, he again reported at the Brigham Hospital. At this time the area of cardiac dulness extended from 2.5 cm. to the right of the midsternal line to 14 cm. to the left. At the apex there was a distinct systolic and a diastolic murmur, and both of these became louder over the base of the heart and along the left sternal margin. It is possible, therefore, that his heart is now slightly larger than it was on his admission to the hospital, but this is not definitely so, for on various occasions during his stay in the hospital the left border was made out to be 13 to 14 cm. to the left of the median line. There is no essential change in the character of the murmurs and, indeed, it is difficult to say that there has been any change in physical signs of the heart since he was discharged from the hospital. With regard to the development of symptoms, he says that he never noticed any shortness of breath until the time he was gassed. Since that time he states that he has been moderately short of breath on exertion. We know, however, that this is not an uncommon result of gassing, and it would hardly be a fair conclusion to regard this as necessarily being a symptom of his heart disease.

One would hardly propose that the physical and nervous stress of war should be used as a functional test for patients with cardiac disease, but in this case the result of an accidental experiment has proved extraordinarily interesting. From the physical examination of his heart few of us would have considered it possible that he could have undergone the strain of the last months. Not many physicians would have been bold enough to advise strenuous exercise for a young man with so large a heart and with aortic and mitral valvular lesions. Indeed, if he had placed himself under a physician's care he would probably, and very properly, have found himself leading a limited

existence. As it is, he has demonstrated again the teachings on which Sir James Mackenzie has so often laid stress. The examination of the heart, and especially the presence of cardiac murmurs, should be regarded as *in themselves* comparatively unimportant matters. The important thing is what the patient can do, and this depends largely on the quality of the heart muscle. Given as strong a heart muscle as this man must necessarily have, and he can compensate for a considerable valvular lesion. A heart with an injured valve works, it is true, at a considerable disadvantage, and its reserve power is usually reduced, but if the muscle is strong and healthy the heart may still do efficient service as long as the valvular lesion is not too extreme. To give a proper prognosis, therefore, we ought to have some knowledge of the condition of the heart muscle. Unfortunately, this is where our methods of physical diagnosis are inadequate. We can determine the condition of the valves with comparative accuracy from our objective examination, but this tells us little about the quality of the myocardium. We still wait for a satisfactory functional test of the heart, and in the meanwhile the best test is to try the heart out under the test of gradually increasing exercise, and see what it can do. Here, then, there is also indicated the path which should be followed in the treatment of the chronic stages of valvular heart disease. The first indication, it appears to me, is to prevent, as far as possible, the occurrence of subsequent infections which may cause further damage to the heart valves and further injury to the heart muscle. The second indication is to increase, as much as possible, the strength of the heart muscle. The problem of increasing the strength of the heart muscle is very similar to the problem of increasing strength of skeletal muscles. Both are improved by properly supervised exercise, which does not reach the point of fatigue. It is an error to believe that all persons with valvular heart disease should lead an extremely limited existence. Too much rest may only result in the production of a weak, flabby heart muscle. Proper exercise will result in the strengthening of the heart muscle so that it may more easily overcome the disability which it suffers from having

injured valves. It is important, of course, that exercise should be somewhat supervised and should not be overdone, for fatigue is of no benefit to cardiac or to skeletal muscle. In general, the best indication as to whether any given exercise is appropriate or not is whether it produces dyspnea, or pain, or palpitation. If exercise causes no distress, it is probably not harmful. Of course, in any given case, it may take some courage on the part of the physician to start his patient on exercise. Much more courage, however, is often needed by the patient, who has usually had it enforced on him that he must be extremely careful about the amount of physical exercise that he takes. If, however, one uses common sense as the guiding principle, the results will rarely be other than beneficial, and the natural outcome is that the cardiac condition will improve as the patient gets into better general training.

The experience which the patient before you has had is by no means unique, and it has been very striking to see how many men with valvular heart disease have stood up under unusual strain in the army. I have personally seen a considerable group of cases of definite aortic insufficiency who have carried on full duty without developing any symptoms, and who have apparently been none the worse off for their experience. The diagnosis was made in these patients as the result of their coming into the hospital for some reason other than heart disease. Many soldiers have, of course, had their hearts give out while in the service, but the majority of these have been older men with myocardial weakness and men with syphilitic lesions. Mitral stenosis, too, seems to have a poorer prognosis than mitral or aortic insufficiency. Not every case can carry on as successfully as the patient just presented to you, but it has been very striking to see how much reserve force the heart may have in a young man with valvular heart disease of rheumatic origin, provided he is otherwise healthy. It is, I think, a profitable lesson for us all to see what these men can stand up under, and it will give us faith that many of our patients in civilian life can probably do a good deal more than most of us would have been inclined to give them credit for. While no one would

knowingly submit such men to the strain which they have been under in the war, many of them seem to have come through without doing themselves any harm.

CASE II.—This patient is twenty-four years old, and before he entered the army he was at work as a clerk. At the age of eighteen he had an attack of rheumatic fever involving the elbows, wrists, and knees. He made a good recovery and was never told that his heart was at all affected. Since that time he has been perfectly well and has been working regularly. He takes a moderate amount of exercise and has never had any symptoms which were referable to his heart. At the beginning of the war he volunteered, but was told by the examiner that he had a "murmur in his heart" and was refused enlistment. Later, however, he was drafted, accepted by the examining board, and sent to one of the large camps. From there he was sent overseas in the spring of 1918, and while in France he "carried on," doing full duty in one of the training areas. After two months he had an attack of acute tonsillitis, and shortly after the onset of this, his right ankle became swollen, red, and tender. He was sent to a hospital, and during his convalescence a doctor again found a murmur on examination of his heart. A diagnosis of valvular heart disease was made and he was transferred through several other hospitals until he was sent back to America. He says that during his stay in the different hospitals there were at several times discussions about his case, and much doubt was expressed by various physicians as to whether or not he had a valvular lesion. He has recently been discharged from the army, and now comes to the hospital for an opinion as to the exact condition of his heart. Subjectively, he apparently feels perfectly well. When questioned directly as to the occurrence of dyspnea, he says that he sometimes gets a little short of breath on climbing two flights of stairs or on going up a comparatively steep hill, but he has been in hospitals for a long time and he thinks that this shortness of breath is accounted for by his being generally "soft." He has had no precordial pain. In appearance he is, as you see, a strong, healthy looking young man and his general physical examina-

tion is entirely negative. Let us now turn to the examination of his heart. The apex impulse is in the left nipple line. The impulse is not unduly forcible and no thrills are felt on palpation over the heart. On percussion, the left border of cardiac dulness is just outside the nipple line, and the right border is 1 cm. to the right of the sternal margin. The action is regular and the heart's rate 80 per minute. On auscultation with the patient standing up the first sound at the apex is of good quality and followed by a soft blowing systolic murmur. This is transmitted a short distance toward the axilla. There is no accentuation of either second sound at the base, but the pulmonic second is somewhat louder than the aortic. On complete expiration there is a rather loud systolic murmur in the second intercostal space just to the left of the sternum. When he lies down the systolic murmur at the apex becomes much louder, and over a small area at the point of maximum impulse the murmur replaces the first sound entirely. The distribution of the murmur is considerably wider when he is lying than when he is standing. The systolic blood-pressure is 125 mm. and the diastolic is 80 mm.

The problem in differential diagnosis before us is to decide whether this is a normal heart or whether the patient has mitral insufficiency. In settling this question, the points in the physical examination to which we must pay particular attention are the size of the heart and the presence of the murmurs. On percussion, the left border of the heart is about 1 cm. outside of the generally accepted normal standard. From this one might be inclined to assume that there is a slight hypertrophy, but, on the other hand, such a percussion outline has been found to be not uncommon in soldiers who are on active duty, and in the early part of the war careful x-ray studies of the hearts of soldiers doing heavy duty in foreign armies showed that slight enlargement was frequently present. The x-ray examination in this case shows that the size of the heart is about at the upper normal limits, and that there is no definite enlargement. The electrocardiogram is perfectly normal and gives us no indication of hypertrophy. In spite, therefore, of the somewhat unusually

wide area of percussion dulness we cannot assume that the heart is definitely enlarged.

Now as to the murmurs. The systolic murmur in the pulmonic area, in the second space just to the left of the sternum, is easily disposed of. It disappears on deep inspiration, but becomes very loud on full inspiration. This is a type of murmur that is very common in normal hearts, and especially in children and in young men and women, but it has been the basis of much discussion in the literature, and the fact that it is not pathologically significant does not seem to be generally appreciated. The exact mechanism by which it is produced is not definitely known, but it seems probable that the disappearance of the murmur on inspiration is due to the fact that the edge of the upper lobe of the left lung becomes interposed between the pulmonary artery and the sternum. Its appearance on full expiration is accounted for by the retraction of the edge of the lung and by the direct pressure of the ribs and sternum on the pulmonary artery. That the occurrence of the murmur is due to pressure on the artery is furthermore suggested by the fact that the murmur is most commonly heard in persons in whom the upper part of the chest is flattened.

The other murmur in this case occurs at the apex in systole and has a blowing character. It is well heard in the erect position, but when the patient lies down on his back it becomes much louder and it is transmitted toward the axilla. In character it is exactly similar to the murmur heard in typical mitral insufficiency. On the other hand, it also resembles very closely the accidental murmurs which are so often heard at the apex of the heart, and of which so much has been said in connection with the examination of soldiers. It is largely since we have been examining such great numbers of normal people that we have begun to find out in how large a percentage of healthy men systolic murmurs may be heard. Owing to a lack of appreciation of this fact the diagnosis of valvular heart disease has undoubtedly been made in a great many instances in which it was unjustified, and it is probable that many men have been kept out of the army for wholly unnecessary cause. The same

problem was apparently met with in England at the beginning of the war, and such authorities as Sir James Mackenzie and Thomas Lewis have laid great stress on the importance of recognizing these accidental murmurs. Indeed, the general feeling of most experts on cardiac diagnosis at the present time is that one should pay very little attention to a systolic murmur in itself, regardless of what its character, transmission, or loudness may be. The importance of a systolic murmur will depend largely on the other features of the cardiac and general examination. Thus, a systolic murmur occurring at the apex in a heart which is otherwise normal should be regarded as having no pathologic significance. On the other hand, a systolic murmur at the apex in association with definite cardiac hypertrophy is of much importance in making the diagnosis of mitral insufficiency.

We have, then, a heart which is not definitely enlarged, and we have a systolic murmur at the apex to which we are agreed importance can only be attached if it is found in association with other evidences of disease. Are we, therefore, to conclude that this heart is perfectly normal and give the patient a clean bill of health? I should be inclined to do so without much hesitation were it not for one important fact in the history. This patient had a typical attack of rheumatic fever at the age of eighteen, and a few months ago he had what appears to have been a second, milder attack. Since we know how very frequently the heart valves and the heart muscles are affected by acute rheumatic fever, it would seem that this point in the history must be regarded with seriousness. In cases of mitral insufficiency, as we usually see them in practice and in general hospitals, the diagnosis is not usually particularly difficult. There is almost always a well-marked hypertrophy of the heart, and frequently a definite accentuation of the pulmonic second sound in addition to the characteristic murmur. The significance of the accentuation of the second sound in the second left intercostal space is often overestimated, for the second sounds at the base vary greatly in intensity in normal persons and with change in position, and also because the sound heard in the so-called "pulmonic area" is often chiefly the aortic sec-

ond sound. Besides these physical signs, however, the patient usually complains of such symptoms as pain in the chest or dyspnea. These are well-marked, clear-cut cases who have usually reported to the physician on account of troublesome symptoms. By the time the symptoms have arisen the diagnosis is usually comparatively easy. We are then dealing with a rather advanced stage of the disease. Is it not probable that there is an earlier stage of the disease in which the diagnosis is much more difficult? This patient—and he represents a large group of men presenting the same problem in the army—did not come to the hospital on account of any symptoms. He feels perfectly well. Is it not quite possible that he represents an earlier stage of the disease than we are accustomed to see? May this not be a stage in which the heart is perfectly well compensated without demonstrable hypertrophy? In the usual course of his life he would never have thought of consulting a doctor at this time, and the findings in his case are to be regarded as practically accidental. If this be the case, then mitral insufficiency, which is usually regarded as being an easy diagnosis to make, becomes an extraordinarily difficult diagnostic problem. It seems to me that one cannot settle the question wholly on the basis of the physical examination. To do so might do the patient a great injustice. The history of attacks of acute rheumatic fever, which are so apt to cause damage to the heart, is too important a factor to be neglected. It would seem to me, therefore, that in a case with this history and with these signs one must incline to consider that we are dealing with an early stage of valvular heart disease.

Having made this tentative diagnosis, one must then be very careful as to the advice which he gives to the patient. The first important point in the treatment of the chronic stage of valvular heart disease of rheumatic origin is to do all that is possible to prevent any further damage to the heart as the result of more acute infections. One should consider the advisability of removing his tonsils, which are rather large, and thus possibly obviate any further attack of rheumatic fever, in case the portal of entry should be the tonsils. We should also caution him as

to the danger of exposure to cold and wet, for it is quite clear that persons who have had rheumatic fever once are more liable to subsequent attacks. It is also fair to tell him that the more frequent attacks of rheumatic fever a person has, the more liable he is to have serious damage done to his heart. The second important point is with regard to the general character of his life. One is often tempted, in such a case, to lay undue stress on the dangers of exercise and overexertion, and to make of him a semi-invalid, whose life centers about his heart. By so doing he is made to lose much of the normal happiness and satisfaction of life, and no adequate benefit is derived as far as his heart is concerned. It is much better to impress on him the fact that he ought to forget his heart and consider only his general physical condition. That which puts him in the best physical training is probably the best thing for his heart. He should be encouraged to lead a healthy normal life, and to take regularly such exercise as he can without any discomfort. He should avoid overdoing, but on the ground that marked fatigue is harmful to the organism as a whole rather than because of its effect on the heart. Beyond this he should be allowed to live a normal life. One should tell him that he has some slight affection of his heart, that it is important for him to return every year to have his heart examined, and that he should report to the doctor if at any time he begins to notice symptoms. He should also be told that he had better come for advice if he plans to undertake any extraordinary amount of exertion, but apart from this he should be told to go ahead and lead a perfectly normal life. For ourselves, as physicians, the important lesson to be learned from this case is that we are confronted with a problem which we cannot definitely solve. On the basis of our physical examination alone we cannot state whether this man's heart has been affected or not. Knowing his history, we must act conservatively, and then we must follow this and other similar cases over long periods of years until we are finally able to determine whether or not there has been any cardiac lesion. When these cases, which have come into our hands in an accidental manner, shall have been followed carefully for long periods,

we shall be in a position to make much more definite statements as to the problem of the early diagnosis of mitral insufficiency.

CASE III.—This patient is an unmarried man of twenty-seven years, who has come to the hospital for an opinion as to the condition of his heart. He says that some years before entering the army he was told that he had "heart disease," but that while he was in the army various doctors told him that this was not the case. His history is as follows: His father has always been a perfectly healthy man, but his mother is extremely nervous and has been so as long as he can remember. He is the youngest of three children, and has one sister who is nervous in the same way that his mother is. As a child he had measles and chickenpox. He has had no other serious diseases, but has never been strong and vigorous. At the age of fourteen, after a short febrile illness, the nature of which he does not know, he was taken to see the doctor, who told him that he had a "weak heart." The only advice given to him was that he must be "very careful." Since that time, he says, he has taken "the best of care of himself." His mother has worried about him a good deal, and has protected him in every eay. He has never been allowed to overexercise. This restricted life has, however, been no particular hardship to him, for he never cared much for active games. Even as a child when he tried to play baseball he used to get tired and out of breath easily. He graduated from high school and then went to work as a clerk in a store. His first job was in a large busy establishment, where the strain was rather heavy, and he found he could not stand it, so he sought out an easier position in a quiet place, where the nervous strain was much less intense. In this new position he has done well, but he gets tired very easily. He rarely loses any time from his work, but he has to be very careful not to overdo. He takes the cars to and from his work and stays at home in the evening. He says he does not exercise, and inquiry reveals the fact that he probably does not walk a mile a day. When he does walk he moves slowly, for he finds that if he hurries he gets tired and out of breath. He is at times troubled with

palpitation, and this may occur without apparent cause at night. He is extremely nervous and is easily upset if any little thing goes wrong. He has occasional headaches. He has found that he cannot stand any unusual nervous or physical strain, and for this reason he has tended to cut down his activities from year to year. In the summer of 1918 he was drafted into the army, and, to his astonishment, he was accepted as a soldier. This surprised him very much, for he says he had always known he had a weak heart. He was started in drilling at the camp, but found much difficulty with it, and says that even on the first days, when the work was light, he became extremely exhausted by it. He noticed palpitation, and then began to feel pain over his heart. When this occurred, he reported to the doctor, but was told that there was nothing serious the matter with him, and that he had better go on with the work. It is rather noteworthy that the amount of physical exercise of the drills at this time was slight, and on direct questioning he states that he was made very nervous by the attitude of the non-commissioned officer. He was much worried by the rigid discipline, and was fearful lest he should do something that would lead his superiors to find fault with him. He was sent one day to the rifle range, but when the firing began he shook so much that the officer took his gun away from him. He says that the shooting "unnerved him." On the way home from the rifle range he felt exhausted and the pain over his heart became intense. He fell out of line on account of shortness of breath, and he says that his heart was beating very fast. He was put into an ambulance and sent to the hospital, where he was kept in a ward until he was discharged, just after the armistice was signed.

On physical examination, you see, he is a fairly well-nourished, rather underdeveloped, nervous looking individual. The color of his lips is good, but both hands are cold, clammy, and cyanotic. When he extends his fingers there is a marked coarse tremor. They thyroid gland is somewhat enlarged and soft. There are no thrills or bruits over it. The general physical examination is entirely negative except for the fact that his reflexes are some-

what hyperactive. The rate of the heart as he stands before you is 125 per minute. The systolic blood-pressure is 140, and the diastolic 90. The apex-beat is very forcible and covers a wide expanse, being seen in several intercostal spaces. There is a slight thrill at the apex, which is apparently systolic in time. On pressure over the apex and in its vicinity, there is definite tenderness. The left border of cardiac dulness by percussion is in the left nipple line, and there is no enlargement of the heart to the right of the sternum. In the erect posture the first sound is loud, and there precedes it a slight roughening suggestive of the presystolic murmur of mitral stenosis. At the base both second sounds are loud. On lying down, the heart is slower. The first sound is less loud and the presystolic roughening has largely disappeared. At the apex a soft blowing systolic murmur is audible. The pulmonic second is somewhat louder than the aortic second. The electrocardiogram is normal and the *x*-ray shows that the heart is not enlarged.

Let us now consider the interpretation of the physical signs. The systolic murmur at the apex, which is brought out by lying down, is quite typical of the common accidental murmurs, and has in itself no significance. There is no hypertrophy of the heart, and there is nothing in the past history to lead us to suspect that there might be a valvular lesion. The presystolic roughening heard in the erect position is of interest because it is a common phenomenon which led to much confusion in the examination of recruits in whom it was often supposed to be evidence of mitral stenosis. It differs, however, from the murmur of mitral stenosis in several ways. It is shorter in duration than the typical murmur of mitral stenosis. It is usually heard in a rapidly acting heart, while in typical mitral stenosis the heart is apt to be slower, unless there is some other complicating factor. The thrill associated with it is systolic in time, and not presystolic, as in mitral stenosis. This murmur is usually best heard in the erect posture, and disappears on lying down, while the murmur of mitral stenosis is more apparent in the recumbent position. The disappearance of the murmur on lying down, when the heart falls away from the chest wall, suggests that the

murmur may be extracardiac in origin. It has been shown by Morison[1] that the two murmurs can be differentiated by an inhalation of amyl nitrite, if one auscults during the period of rapid heart action. Following the inhalation one finds that the murmur of mitral stenosis becomes louder, while this spurious murmur disappears. There is, indeed, a certain resemblance between this murmur and that of mitral stenosis, but if one bears in mind that this type of sound is common in the overacting hearts of nervous people with thin chest walls, there should be little difficulty in distinguishing the condition. In the absence of any hypertrophy, then, and in the absence of a history of rheumatic fever, we can safely exclude the presence of valvular heart disease in this case. We must consider the possibility of the tachycardia being due to Graves' disease, and in many similar cases in the army this diagnosis has been made. He has, it is true, a somewhat enlarged thyroid gland, and he comes from the region of the Great Lakes, where it is well known that goiter is endemic. The thyroid gland is, however, not larger than is frequently found in young adults, and it is softer than is commonly the case in Graves' disease. Moreover, it has been found that if he lies down quietly for an hour or so his pulse-rate drops to 80. This is an important point in the differentiation, for in Graves' disease the pulse does not usually show such complete slowing with rest. A good deal of investigation has been made into the problem as to whether cases similar to this depend on overactivity of the thyroid gland. In England Dr. Thomas Lewis found that the administration of thyroid extract did not increase the symptoms, and in this country, at General Hospital No. 9, in Lakewood, New Jersey, it was found that the basal metabolism is normal. The evidence is, therefore, entirely against the fact that the thyroid gland bears any relation to this condition. Clinically, also, the patients are of a different type, for while they are often extremely nervous, they lack the characteristic energy and "drive" of the typical hyperthyroid subject. It is usually extremely difficult to make a soldier with Graves' disease undergo a rest cure in bed, but I feel quite cer-

[1] Morison, J. A., Brit. Med. Jour., i, 1918, 452.

tain that we should not have any difficulty in enforcing such a quiet, dull régime on this man.

This patient presents the combination of nervous and physical symptoms, which are typical of the condition generally known as the "irritable heart of soldiers" or "effort syndrome." A tendency to fatigue easily is perhaps the most characteristic feature of the picture. With this one finds dyspnea on slight exertion, and palpitation and precordial pain coming on even when at rest. A rapid pulse, a coarse tremor, involving the fingers, hands, arms, and sometimes the legs, cyanosis of the extremities, and marked sweating are common accompaniments. Associated with this, and often overshadowing everything, is a very neurotic temperament. On account of the large number of recruits presenting this clinical complex, the condition became one of great importance in the army, but it is also a condition which is very common in civil life. One may recognize in general two distinct types. In the first place, there are those in whom the symptoms are of relatively short duration. In these men the symptoms have arisen in the army, and they are directly or indirectly the products of army life. The commonest etiologic factors seem to be infection, gassing, and the nervous and physical strain of warfare. In many instances the patients in this category are analogus to those in whom the diagnosis of "shell-shock" is made, and, indeed, many of these men are sent to neurologic hospitals. It often seems to be purely a matter of chance as to whether they are sent to a cardiac ward or a neurologic ward. Cases similar to these, in which the symptoms can be traced to some psychic trauma, are also seen in civil life, but they are less frequent than the second group of patients. In this second group the symptoms are of long standing and often date back to childhood. It is of this type that the patient before you is an example. In them inheritance may play a rôle, for it is not uncommon to find some nervous factors in the family history. The subjects of this group cannot be regarded as being directly the product of the war. Their symptoms have existed for many years before they entered the army, but they were able to limit the production of their symp-

toms by living below the level of nervous and physical strain which brings them out. This was the commonest type of "effort syndrome" seen in our army, but it is probable that if the war had continued longer, and our men had been forced to go through the severe strains endured by our Allies, we should have had many more of the acute cases. Before entering the army these men have usually been regarded as underdeveloped normals, or perhaps they have been classed as neurasthenics. In general, they have got along pretty well because they have been able to adapt their lives to their own limitations. They have contented themselves with living on a low physical and economic level. When, however, they are called to the army, it becomes necessary for them to adapt themselves to a new life—a life that is full of physical and nervous strain. It is then that they fail to come up to requirements, and that they appear in the light of pathologic subjects. In spite of the fact that our attention has been only directly called to the problem during the mobilization of the army, it is quite evident that the same problem exists in civil life and that it should be recognized and treated.

The various types of "effort syndrome" present a variety of clinical pictures. Underlying these long-standing or constitutional cases there are both physical and nervous elements. The importance of the physical element was brought out at General Hospital No. 9, at which it was shown that the muscular strength of many of these men is well below the average. Indeed, special tests showed that in a considerable proportion the general muscular condition was extremely poor. They had never taken much exercise, and had never been in good training. They were very "soft." One could hardly expect that they could stand up under any severe physical strain, for they had led for many years a sedentary life, and had devoted much energy to the avoidance of all physical exercise. It would, of course, take a long time to develop them to a point where they could march with full equipment. But the physical element is not the only factor of importance; there is also a large nervous element. Most of these men are very neurotic. They worry, are anxious,

and are easily upset. The man who has been presented to you told us that one of the things that bothered him most in his drilling was his anxiety lest the sergeant should find fault with him. They almost always have a great fear of "overdoing." They know from experience that when they overdo they "feel badly," and they fear that something serious may be about to happen to them. Unfortunately, the physician is not infrequently an important element of the production of this phobia. In a very large proportion of cases a doctor has said that they had a "weak heart," and has told them to do as little as possible. An anxious family has become worried about them, and has, in turn, done everything possible to limit their activities and to protect them from every kind of activity. Their lives center about their hearts. They are continually cutting down their activities. Instead of developing themselves physically, they have done exactly the opposite. Were the advice given by the doctor based on a proper comprehension of the case, the treatment might well be justified, but too often the diagnosis of "weak heart" shows that the clinical condition has been totally misunderstood. If there is disease of the heart, it is, except in very unusual cases, essentially disease either of the valves or the muscle, and in young persons the former is much the more common. It is probable that the diagnosis of valvular heart disease has frequently been made in the past when it did not exist, but as a result of our experience in examining large numbers of healthy men in the army, the profession as a whole has learned much about the presence of "accidental systolic murmurs," and doubtful presystolic rolls in normal hearts. Errors in the diagnosis of valvular disease should be much less common in the future. Myocardial disease, unaccompanied by valvular disease, on the other hand, is rarely met with in young people except as a result of syphilis, in association with hyperthyroidism or perhaps immediately after severe acute infections. In conditions other than these, in young persons, the diagnosis of "myocarditis" is open to doubt. In the type of case which we have under discussion it seemed of prime importance to determine whether there might not be some true myocardial involve-

ment, but investigations made in this country and in England have failed to show any definite evidence that this is the case. As far as the heart goes, the most that can be said is that there is an upset of the balance of the cardiac nerves. Whether this is principally due to an increased activity of the sympathetic system or a decrease of the vagus control is not at present certain. The exact mechanism by which the heart is affected so that it responds to slight exertion, or to nervous excitement by an abnormal increase in rate, is not at all clear, but the essential thing for us as practitioners is that we should recognize more frequently the so-called "irritable heart" in civil life, and should appreciate the fact that the cardiac element is purely secondary. The fundamental feature is probably a neurosis, and associated with this there is often a lack of general physical development. To lay stress on the heart in these cases is to create a semi-invalid. Experience has shown that it is much easier to direct a person's attention toward his heart than it is subsequently to draw his attention away from his heart, and the mobilization of our army has demonstrated how large a group of persons there are who are going through life, losing much of its joy and usefulness because of a heart disease that does not exist. The seriousness of an error of diagnosis in this type of patient can hardly be overestimated, for the neurotic, introspective temperament causes exaggerated anxiety, and the fear of injuring himself further results in the leading of a miserable existence. First, then, there must be a correct diagnosis. This will then lead to proper therapeutic measures. The physical aspect of the case will be treated by gradually increasing physical exercise and directions as to the leading of a normal healthy life. The nervous element is more difficult to handle. Encouragement, the instillation of self-reliance and self-respect, the direction of the attention away from bodily ailments and general psychotherapeutic methods are the essentials. But back of it all there lies too often the inherited neuropathic constitution, and one is reminded of the old saying that the most important thing for a child is the wise choice of his parents.

CLINIC OF DR. GEORGE CHEEVER SHATTUCK

MASSACHUSETTS GENERAL HOSPITAL

CASES FROM THE MALE MEDICAL CLINIC OF THE OUT-PATIENT DEPARTMENT

Case I.—Chronic Pulmonary Tuberculosis and Arteriosclerosis.

Case II.—War Nephritis and Chronic Adhesive Mediastinopericarditis Probable.

Case III.—Syphilis, Lesion of Aortic Arch, Probably Syphilitic; Healed Ulcer of Stomach or Duodenum.

April 9, 1919.

CASE I.—CHRONIC PULMONARY TUBERCULOSIS AND ARTERIOSCLEROSIS

No. 383,010—Frank W. Born in Connecticut, living in Boston. Age sixty-five, white. Occupation, bootmaker for twenty-five years, janitor for six years.

Present Illness.—The patient says that he has had coughs and colds every winter since a boy, that he coughs less in summer, raises about a tablespoonful of sputum, which is often thick and yellowish, but variable in appearance, and that he has never spit up blood. He has had no pain in the chest except occasionally with "indigestion not influenced by exertion."[1]

There has been gradual loss of weight for ten or twelve years aggregating about 40 pounds. For the same period there has been moderate dyspnea on exertion.

Past History.—Unimportant except as above stated.

[1] When a patient over forty years of age complains of "indigestion" be sure to exclude angina pectoris by determining whether the pain is brought on by exertion.

Family History.—Father died of "old age." Mother had *severe cough during the later years of her life.* A brother died suddenly with pain in the chest and shortness of breath. A sister died of "heart trouble." No tuberculosis or malignant disease known in the family.

Physical Examination.—Well developed and fairly well nourished. Brownish pigmentation across forehead irregular in outline. Pupils equal and react to light and accommodation. Knee-jerks present. Nasopharyngeal catarrh. Hoarseness.

Heart dulness not increased. No murmurs. Chest symmetrical, no shrinkage at apices. Lungs hyperresonant in front, slight dulness above right clavicle, breath sounds not definitely abnormal. Coarse râles at right apex in front, at both apices behind, and at left base. Abdomen negative.

Temperature 98.8° F., pulse 74, blood-pressure, systolic 190, diastolic 100. Urine, color normal, clear, acid; specific gravity 1018; no albumin or sugar.

DR. SHATTUCK: What is your diagnosis?

STUDENT: Chronic bronchitis.

DR. SHATTUCK: The physical examination does not warrant you in stating more, but that diagnosis should not be accepted as final. What are the three conditions which often pass under the name of chronic bronchitis?

STUDENT: Tuberculosis.

DR. SHATTUCK: That is the most important and commonest one. Can you mention another?

STUDENT: Chronic cardiac insufficiency.

DR. SHATTUCK: How does that resemble bronchitis?

STUDENT: There is chronic passive congestion and sogginess of the lungs.

DR. SHATTUCK: In such cases where are the râles most numerous?

STUDENT: At the bases of the lungs.

DR. SHATTUCK: Do you think that cardiac insufficiency is a factor in this case?

STUDENT: No. The râles are most numerous in the upper parts of the lungs.

DR. SHATTUCK: What is the third condition, often called "chronic bronchitis"?

STUDENT: Syphilis.

DR. SHATTUCK: That also should not be forgotten in cases in which the diagnosis is obscure, but it is not the disease I had in mind. I was thinking of bronchiectasis. Bronchiectasis is characterized by daily expectoration of purulent sputum varying in different cases from a teaspoonful to a cupful, generally more abundant in winter than in summer. It is a sequel of influenza and perhaps of bronchopneumonia independent of influenza. It runs an extremely chronic course, shows no marked tendency to shorten life, and interferes little with nutrition. In this respect it differs markedly from tuberculosis, in which, as you know, loss of weight is generally but not always a feature. In this connection I recall an autopsy on an old man who had extensive cardiac disease and tuberculosis for years. His death was due to lobar pneumonia, but active tuberculosis of unexpected extent was found. There was much fibrous walling off and caseation, showing the chronicity and slow extension of the process. During life, aside from dyspnea on the least exertion, the man was the picture of health. His skin was clear, color good, and nutrition excellent. Bronchiectasis differs again from tuberculosis in being generally confined to the bases. It may be unilateral or bilateral, may honeycomb and destroy large areas of lung tissue, or may be limited to a few small foci, showing on physical examination only a few râles. It is these slight cases which are generally called chronic bronchitis, but the more extensive ones often drift to sanatoria for tuberculosis in spite of repeated negative sputum examinations. In tuberculosis with abundant sputa tubercle bacilli should be demonstrable. It is the early case in which their absence need not excite surprise. Another feature of bronchiectasis which is important is the comparative absence of constitutional disturbance manifested by tachycordia, fever, and loss of weight, all which are generally present in tuberculosis. Its other manifestations are not related to the case under discussion. In our case the signs are in the

upper part of the lungs and not at the bases. This is strong evidence against bronchiectasis.

In favor of tuberculosis we have evidence of chronic pulmonary disease most marked at the apices and associated with constitutional disturbance, namely, marked loss of weight. The dulness at the right apex seems more than physiologic, but the absence of external shrinkage is noteworthy. Extensive dulness may be masked by the hyperresonance of emphysema. The normal pulse and temperature are not significant in a man of sixty-five, a time of life in which tuberculosis generally runs a slow course with little fever. Moreover, these are single observations made in the morning.

In summing up it may be said that tuberculosis should be suspected in every case of so-called chronic bronchitis, that in elderly persons it often presents atypical signs marked by emphysema, that the symptoms may be so slight and the onset so gradual as to escape the serious attention of the patient for many years.

The evidence in this case points first to tuberculosis. If this diagnosis cannot be confirmed, we will go over the ground again.

In addition, the patient has peripheral arteriosclerosis and probably renal arteriosclerosis. The high blood-pressure suggests the latter, and absence of albumin at a single examination does not exclude it. Although cardiac dulness is not increased, the heart is almost certainly hypertrophied. Palpation of the apex impulse in the left lateral position would probably show this. What further evidence is needed to establish a diagnisos?

STUDENT: Examination of the sputum.

DR. SHATTUCK: Yes, we will have an *x*-ray plate, too, to see what that will show, and I should like to have the larynx examined.

Second Visit (April 12th—three days after first visit).—Many medium moist râles at apices, more on the right than on the left. Few râles lower down. Bases clear. Sputum examination: Tubercle bacilli found.

x-Ray Report (by Dr. Holmes).—"Mottled dulness at both

apices to second rib in front on the right. General thickening and mottling of lung markings throughout the chest. Diaphragm excursion limited on both sides. Aortic arch tortuous and prominent." Interpretation: "Old tuberculosis at both apices, arteriosclerotic dilatation of aortic arch."

Throat Examination (by Dr. Barnes).—"No enlargement of arytenoids. Vocal cords roughened."

Final Diagnosis.—Advanced phthisis, arteriosclerosis.

Disposal.—Referred to Social Service for advice about hygiene and to arrange for sanatorium treatment to protect public.

April 21, 1919.

CASE II.—WAR NEPHRITIS AND CHRONIC ADHESIVE MEDIASTINO-PERICARDITIS PROBABLE

No. 383,784—Jean L. Born in Canada, has lived most of his life in the United States. Age nineteen. White. Discharged soldier in Canadian Infantry.

Present Illness.—Captured by Germans on September 6, 1918, and sent at once to working camp, where he helped to build dug-outs. In the camp he was cold and wet most of the time. About September 15th he noticed swelling of the face, soon followed by edema of the ankles, which increased gradually. On September 23d he was relieved of work, but the swelling continued to increase. About the end of September he was sent to a hospital, where he spent two or three weeks. He was then transferred to another hospital in Antwerp, where he stayed ten days. From there he was sent to a prison camp, where there was a hut with stoves in it for the sick. While there his "stomach swelled way out." There was no dyspnea even at first.

On December 12th he was sent to a Canadian General Hospital at Boulogne, where for the first time he received systematic treatment and began to improve. His abdomen was tapped three times. On the 16th of December 3600 c.c. of clear watery fluid were withdrawn and on the 18th 7800 c.c., according to

notes forwarded with the patient. The notes continue as follows:

Dec. 16, '18: "Bl. p. syst., 188; diast., 124."
Jan. 1, '19: "Looking ill, marked dropsy, double detached retina."
Feb. 20th: "Feeling all right. Slight edema of shins and ankles. Was blind for three weeks. Now says there is a haze over eyes and can see only in brightest light. Pulse good and slow. Fluid still present in abdomen. Patient looks white and anemic."
"Urine: albumin + +, pus and hyaline casts."
March 6th: "Alb. retinitis, vision less than 6/60."
"Urine: pus, hyaline and granular casts about 40–50 ounces.
"The patient says that sight is steadily improving, that he sees well now, that his appetite is excellent and that he feels better in every way."

Past History.—Diphtheria and measles when a small child, tonsillectomy at age of ten; no scarlet fever or rheumatic fever.

Physical Examination (April 21, 1919).—Well developed and fairly well nourished. Skin and mucous membranes of good color. Pupils equal and react to light and accommodation. Albuminuric retinitis, slight. Throat negative. Several carious teeth. Tongue coated.

Heart.—Apex impulse visible and palpable in fourth space within nipple line. Dulness slightly increased to right and left. Action regular and not rapid. Aortic second sound much accentuated and ringing. After slight exertion a faint systolic murmur is heard at the apex. In the left lateral position the apex impulse is felt a little beyond the anterior axillary line. The force and width of the impulse are increased. Blood-pressure: Syst., 185; diast., 135.

Lungs and *abdomen* negative. Knee-jerks exaggerated. The skin of the abdomen, thighs, and knees shows numerous and well-marked lineæ albicantes. No edema.

Urine: Cloudy, specific gravity 1019, albumen ++, many leukocytes and granular casts.

Temperature 99° F. Pulse 72. Weight 144 pounds.

Second Examination (April 23d).—Slight systolic retraction of chest wall below nipple and over tenth and eleventh ribs in

posterior axillary line. Pulsus paradoxus slight in degree. In recumbent position soft systolic murmurs are heard in the pulmonic area and in the mitral area, not transmitted to the axilla. There is also a short diastolic murmur heard near the mitral area.

Aortic second sound loud and ringing as before. Pulmonic second sound also accentuated. Blood-pressure: Syst., 165; diast., 110.

DR. SHATTUCK: What is the diagnosis?

STUDENT: Nephritis.

DR. SHATTUCK: Why is the apex in the fourth interspace?

STUDENT: Perhaps the heart was pushed up by the ascites and became adherent.

DR. SHATTUCK: Does the heart move with change of position?

STUDENT: Yes. The apex impulse moves to the left when the patient lies on the side.

DR. SHATTUCK: The cardiac dulness on the right shifts also, so that the conclusion seems justified that adhesions if present are not very extensive, but there might, I think, be some adhesions nevertheless. In favor of their existence is the retraction of the chest wall and ribs near the apex of the heart, and in the back.[1] The pulsus paradoxus points the same way, but is of little diagnostic value. The pronounced ascites is very unusual in a renal case in which the heart has not been overtaxed. In this case there is no reason to believe that the heart was a factor in the dropsy. I do not remember having ever seen paracentesis done for ascites in uncomplicated nephritis, but in this case large amounts of fluid were thus removed, and tapping was required three times. The prominence of the ascites in the clinical picture suggests chronic adhesive mediastinopericarditis as a possible cause, and the signs of adhesions above referred to strengthen this hypothesis.

The force and width of the apex impulse as felt in the left lateral position point to hypertrophy, probably moderate in

[1] A very much hypertrophied heart without adhesions may cause retraction near its apex, but in this case hypertrophy is slight.

degree. If there were dilatation of more than a slight degree the apex would be farther out or farther down. The increase of blood-pressure three times recorded is consistent with hypertrophy. The murmurs next require explanation. The answer is not clear to me. The systolic murmurs in the pulmonic and mitral areas may be the same and may be functional or due to slight relative insufficiency of the mitral valve. Perhaps the systolic murmur at the base is produced at the aortic valve, but if so it is heard in an unusual position. It seems probable that the diastolic murmur is of aortic origin, for it is not of the presystolic type nor is there any accentuation of the first sound to suggest mitral stenosis.

On the whole, it seems probable that there is a well-compensated lesion of the aortic valve of slight degree and perhaps of the mitral as well. The lesion may have developed in childhood as a result of infection from the tonsils which were removed, or it may be associated with the nephritis, as heart lesions often are in the glomerulonephritis of civil life. I do not remember to have seen a heart lesion develop in a case of war nephritis.

The diagnosis, in brief, is:

1. Subacute glomerulonephritis beginning under war conditions.
2. Hypertrophy of the heart with little or no dilatation.
3. Chronic endocarditis of the aortic valve, slight in degree, seems probable.
4. Albuminuric retinits.
5. Chronic adhesive mediastinopericarditis seems probable.

War nephritis is an acute nephritis. In civil life acute nephritis is mainly a disease of childhood, but acute exacerbations of chronic glomerulonephritis in the adult are common and may simulate acute nephritis. In war nephritis the kidney lesions are very like those of acute glomerulonephritis. Acute glomerulonephritis has been shown to be of bacterial origin, so that by analogy war nephritis should be of similar origin, but proof is lacking. Having seen war nephritis common in the spring, scarce in the summer, and recurring in greater numbers again

in the autumn of three successive years in France, I am convinced that exposure to cold and wet is a factor in the great majority of cases. I believe also that infection plays the decisive part.

The absence of dyspnea at the outset in this case is strikingly unusual, for the patient was doing heavy work. A common story is that the patient became short of breath while marching with equipment, that the dyspnea increased from day to day until he was obliged to fall out, and that later edema of the ankles or face was noticed.

The *prognosis* is uncertain. The mortality in the acute stage of war nephritis is very low. Uremic symptoms when they occur generally yield readily to treatment. Undoubtedly a certain proportion of cases become chronic and will die ultimately of the disease, but what this proportion is cannot yet be known. The rapid improvement of most cases offers hope of cure in a large proportion. In this case the pus in the urine and the duration of the illness seem to me unfavorable signs, and I think the case is passing into the chronic stage.

Treatment requires limitation of foods, the waste of which is mainly eliminated by the kidney. Water need not be restricted in this case, for the heart is strong and the absence of edema shows ability of the kidney to excrete water.

Salt need not be forbidden entirely, although it might be unwise for the patient to take much of it. We may tell the patient to use salt sparingly, but a strictly salt-free diet is quite unnecessary.

The patient is gaining in weight and strength and shows little sign of anemia now. It would be a mistake to put him on a much restricted or monotonous diet, of which he would soon grow tired, because he would be unable to eat enough to maintain himself in good general condition. Now that the acute stage has passed and the appetite has returned, the patient may eat a little meat or fish once daily and eggs need not be restricted. Starchy foods and vegetables of any kind can be eaten freely. Acids, highly spiced foods, meat broths, and alcohol should be avoided.

Drugs are not needed at present. Exposure to cold must be avoided. Woolen underclothing should be worn. Physical exertion should be moderate. If appetite fails or if headache or edema should be noticed, the patient should at once restrict the diet and return to the hospital. In any case, he is to report once a month.

March 31, 1919.

CASE III.—SYPHILIS, LESION OF AORTIC ARCH, PROBABLY SYPHILITIC; HEALED ULCER OF STOMACH OR DUODENUM

No. 382,260—William P. Age fifty-nine. White. Married thirty-seven years. Occupation, teamster for forty years.

Family History.—Wife and 5 children living and well. One child died of pneumonia and another shortly after birth, cause unknown. There were no miscarriages.

Past History.—Says he has had indigestion for ten or fifteen years, with epigastric pain from one to two hours after meals, and sometimes keeping him awake until midnight. No sharp attacks like biliary colic. Vomiting has been frequently associated with the pain. Vomitus sometimes watery and sometimes containing much food. Ten years ago he vomited a pint of blood, but this has not recurred. Stomach trouble now is slight.

Thinks he had syphilis twenty years ago and had received little treatment for it. Nocturia two or three times for years.

Present Illness.—Dyspnea on exertion for six months, but never associated with pain, he thinks. Has been doing heavy lifting, but aside from moderate dyspnea, which soon passes off, has observed no ill effect.

Physical Examination.—Well developed, obese. Pupils small, equal, and react normally. Teeth poor. Throat negative.

Heart.—No clearly demonstrable enlargement. Action not rapid, but when first examined there was an irregularity recurring with every fourth beat. Half an hour later only an occasional premature systole was heard. No murmurs were heard. Blood-pressure: systolic 150, diastolic 120.

Lungs negative. *Abdomen* negative.

Knee-jerks sluggish. Varicose veins on both legs and moderate edema to knees. Temperature 98.6° F. Weight 235 pounds.

DR. SHATTUCK: What do you think is the matter with this patient?

STUDENT: Duodenal ulcer.

DR. SHATTUCK: Why has he edema of the legs?

STUDENT: That is probably due to the varicose veins.

DR. SHATTUCK: The past history is strongly suggestive of gastric or duodenal ulcer. The type of pain, the duration of symptoms, and the vomiting of blood all point to this conclusion, but the symptom which seems to me of chief importance now is the dyspnea. Although the heart shows little evidence of enlargement, the chest wall is so thick as to render percussion valueless. I should not be willing to say that the heart is normal without further evidence. The premature systoles in a man of fifty-nine years of age are more significant than they would be in a young man, and in this case they may indicate myocardial changes of an important kind. Moreover, the edema of the legs may not all be due to the veins. Before making a final diagnosis we must have more evidence, but I believe that the heart is beginning to weaken and that the patient should be treated accordingly. The recognition of slight signs of failing compensation is very important, for in such cases much good may often be accomplished.

There is another question which merits consideration at this time. If it be assumed that the myocardium is weak, what is the etiology? Physically the patient is not senile nor does he show the marked degree of peripheral arteriosclerosis so common in senility. The past history gives one important clue, namely, that the patient thinks he had syphilis twenty-one years ago. The family history is non-committal, but evidence of active syphilis should be looked for in this case nevertheless.

We shall want *x*-rays of the heart and aorta and of the gastro-intestinal tract and a Wassermann test.

Treatment.—Epsom salts, 2 tablespoonfuls[1] every other day before breakfast, and tincture of digitalis, min. x t. i. d.

April 1st: Wassermann test strongly positive. *x*-Ray report by Dr. Holmes: "Well-marked evidence of hypertrophy of left ventricle. Shadow of great vessels increased. Gastro-intestinal tract negative."

April 3d: Second visit. Iodid and mercury prescribed.

April 15th: Third visit. Feels much better. Dyspnea less. indigestion slight. Pulse not rapid. Occasional premature systole. Blood-pressure 145 systolic and 100 diastolic. After climbing two flights of stairs pulse 108.

Heart: Sounds distinct, none accentuated. No murmurs heard in the sitting position. When the patient lay on his back a faint systolic murmur was heard in the aortic area when the lungs were deflated and the breath held. In the left lateral position the apex impulse was felt in the anterior axillary line and a soft systolic murmur was heard at the apex.

Referred to South Medical Department for treatment of syphilis. They administered 0.1 gm. diarsenol intravenously.

Further Discussion of Case.—When a heart is much enlarged the apex impulse, as a general rule, can be felt in the fifth or sixth space in the *midaxillary* line when the patient lies on his left side.

In this case the impulse moved only to the *anterior* axillary line and it was not noticeably increased in force. I think, therefore, that the enlargement of the heart suspected at the first examination and reported by the radiologist is slight in degree. The heart is not notably dilated.

The murmur at the aortic area indicates a slight lesion of the valve or of the arch. The arch has been shown to be slightly enlarged, but the enlargement is not of the type characteristic of syphilis in that it is not limited to the first part of the aorta. If it were so limited, syphilis of the arch at an early stage would be extremely probable from that evidence alone.

[1] The large dose of salts will help to reduce weight, which is essential in obesity associated with cardiac weakness.

The murmur was probably overlooked at the first examination. These incipient murmurs are generally overlooked unless sought for with the greatest care. When syphilis involving the heart or aorta is suspected, systolic and diastolic murmurs should be looked for most carefully. Deflation of the lungs and examination in the left lateral position will reveal many murmurs that would not otherwise be found. When found, they increase the probability of syphilis as a factor, unless they can better be explained on some other hypothesis. They are less important in senility, because at this period of life they are commonly the result of degenerative changes. When such a murmur is found before the patient has reached the age of thirty it is generally attributable to an endocarditis of bacterial origin. The possibility of syphilis as a factor in the case should not be forgotten either in the young adult or in the aged, but when a heart lesion is first discovered between the ages of thirty-five and fifty the probability, *a priori*, is in favor of syphilis as the cause. This fact cannot be too strongly emphasized because early diagnosis gives hope of good results from treatment, but after the damage has become extensive no treatment can cure, and the prognosis in such cases is grave even with the best treatment.

Another interesting possibility exists in this case. Might the stomach trouble also have been due to syphilis? Cases of ulcer with hematemesis have been attributed to syphilis more and more frequently in recent years. Whatever the cause in this case, the lesion seems to have got well without leaving any signs discoverable by *x*-ray examination.

April 24th: Fourth visit. Patient feels about the same. Slight dyspnea persists. Less edema of legs. Left leg swells occasionally. Pulse-rate at rest 84. After climbing two flights of stairs quickly it reached 124 and showed occasional irregularity. Diarsenol 0.1 gm. administered by South Medical Department.

Final Diagnosis.—Syphilis, lesion of aortic arch, probably syphilitic. Healed ulcer of stomach or duodenum, perhaps syphilitic.

Disposal.—Undergoing treatment.

CONTRIBUTION BY DR. ARIAL W. GEORGE
AND DR. RALPH D. LEONARD

THE USE OF THE *x*-RAY IN THE STUDY OF MULTIPLE DIVERTICULITIS OF THE COLON

Recognition of Diverticulitis as a Clinical Entity; Constipation as an Etiologic Factor; Pathologic Changes; Symptoms; Differential Diagnosis; Radiographic Classification of Multiple Diverticula; Bibliography.

THE term "multiple diverticulitis," when applied to the large intestine, refers to the presence of one or more sacculations projecting from the exterior of the gut, together with their secondary inflammatory changes. As many of these pathologic sequelæ involve the tissues around the intestine, "peridiverticulitis" might be a more apt term.

Diverticulitis today occupies an important place in medical literature and is recognized as a distant clinical entity by every operating surgeon of large practice. It is only within the past twelve or fifteen years that the profession has had an intelligent appreciation of the importance of this condition, although Graser presented in 1898 a fairly accurate clinical and pathologic description of "diverticulum formation" in the large bowel and showed that such cases were not uncommon.

Previous to Graser's work, however, the literature on "diverticulitis" consisted in the occasional report of isolated cases. Virchow in 1853 described certain "pathologic changes involving the descending colon and sigmoid characterized by isolated, circumscribed, adhesive peritonitis." He even described some of the possible complications, adhesions, constrictions, perforation, etc. He did not, however, note the presence of diverticuli

as the original cause, considering constipation as the etiologic factor, neither did he attempt to describe the clinical picture.

From 1900 on many important contributions were offered, particularly by American observers. Fisher in 1901 and Beer in 1904 did experimental work on the etiology of the intestinal diverticula. In 1907 the Mayos, Wilson, and Griffin reported several operated cases of diverticulitis, contributing particularly to the clinical aspects of the disease. In the same year Brewer offered a paper also describing the clinical manifestations of diverticulitis entitled "Etiology of Certain Cases of Left-sided Intra-abdominal Suppuration." In the following year Ashhurst reported a case of "Sigmoid Diverticulitis" in a child. This patient is the youngest case on record. McWilliams about the same time reported cases emphasizing some of the serious sequelæ.

Telling in 1908 for the first time collected and analyzed the recorded cases, classifying the pathologic changes and clinical results. This classification has furnished the basis for most articles of importance written since that date. In 1911 Telling and Gruner amplified this classification, basing their conclusions on a much larger number of cases.

During the last seven or eight years there have been many contributors to both the clinical and pathologic features of "multiple diverticulitis," among whom are Mayo, Wilson, Giffin, Hartwell and Cecil, Graves, Erdmann, and particularly McGrath. His paper on the "Etiology and Pathogenesis of Colon Diverticula" is particularly valuable.

The *x*-ray as a means of diagnosis of multiple diverticulitis was first brought to our attention by Dr. Stewart, who made a Roentgen diagnosis of sigmoid diverticulitis in a case reported by Abbe in August, 1914.

Carman, in November of the same year, and Case, in the following year, described the radiographic appearance of multiple diverticulitis of the colon. Since then a few contributions on this subject have been made by roentgenologists to periodicals and text-books. These papers, however, are in the nature of

reviews and confirmation of Carman and Case's work, and offer nothing particularly new.

In order to better understand the radiographic appearance of multiple diverticulitis it may be wise to consider some of the pathologic aspects. The following statements and classifications represent in general a résumé of the more recent contributions to the pathology and etiology of this disease.

Diverticula may be classified in two general groups—the congenital group and the acquired group. The congenital group is small. Meckel's diverticulum in the small intestine and a few other developmental anomalies, such as a persistent urachus, compose this class. These congenital diverticula do not enter into the scope of the present paper.

As Dr. Charles Mayo states, "The various hollow viscera and tubular structures of the body are subject to acquired diverticula. Even the large blood-vessels are prone to this condition in the form of sacculated aneurysm."

The group of "acquired diverticula" comprise practically all the diverticula which are of clinical importance. The esophageal diverticula, the various diverticula of the duodenum, ileum, and the multiple diverticula of the large intestine, in which we are particularly interested, belong to this group.

The acquired diverticula are further described as "true" or "false," these terms being used with reference to the structure of the walls of the diverticulum, as compared to the structure of the walls of the normal organ. Some pathologists use the terms "complete" and "incomplete" synonymously with "true" and "false."

A "complete" diverticulum presents in its walls the same tissues and same arrangement of lining membranes as the normal intestinal wall. The "incomplete" or "false" diverticula consist of but part of the elements found in the normal intestinal wall. The diverticula of the colon probably belong to this latter group, their walls being composed of merely the mucous and serous coats. There is no "muscularis" as found in the normal intestinal wall. McGrath speaks of these diverticula as "hernia mucosæ."

It has been suggested that originally they were "true' diverticula, the muscle coat simply atrophying from disuse. In view of the probable etiology of diverticula we feel that the idea of "muscle atrophy" is rather unlikely.

Diverticula of the large bowel may be found in any division of the large intestine, but are most commonly found in the descending colon and sigmoid. McGrath's figures in a series of 32 cases are typical. In 27 cases diverticula were found in the descending colon and sigmoid, 2 cases in the rectum, 1 case in the transverse and hepatic flexure, and 1 in the anal ring.

Our observations as radiologists would lead us to believe that diverticula occur in the ascending and transverse colons more frequently than has generally been believed. The diverticula in this location, however, are less likely to give symptoms than in the descending colon and sigmoid, hence do not come to the attention of the surgeon or pathologist.

The number of diverticula present may vary from 1 to 100 or more. Hauseman found 400 at autopsy in a man of eighty-five dying from pneumonia. It is generally agreed that colon diverticula are multiple and that 10 to 20 are the average number found.

They vary in size from a small fraction to 2 or more inches in diameter. The tendency is to gradually increase in size, the earliest stages being microscopic. The average size found at autopsy or operation is about that of a pea.

They are of variable shape, being usually round or ovoid. Some are distinctly pedunculated, with a minute opening into the lumen of the intestine.

The contents of these pockets is almost entirely fecal material, the consistency of which is variable. Occasionally the fecal material becomes so inspissated as to produce fecaliths. McGrath speaks of "concretions becoming incapsulated within the diverticula and, the pedicles sloughing, give rise to free bodies in the abdominal cavity." Numerous writers have reported foreign bodies in the diverticula.

The diverticula project from the exterior of the gut, usually close to the mesenteric attachment (Figs. 254 and 255). They

may be found between the two layers of peritoneum forming the mesentery. It is also observed that the diverticula are in close proximity to the epiploic appendices.

Under the microscope the walls of the diverticula are seen to consist of mucosa, usually submucosa and serosa. There is

Fig. 254.—Diverticula of sigmoid. The pouches are, for the most part, into the appendices epiploicæ. (Taken from Maxwell Telling and Gruner, British Journal of Surg., vol. iv, No. 15.)

complete absence of smooth muscle-fiber. In some of the larger diverticula the lining of epithelium is more or less obliterated, due to the continued pressure of the fecal contents.

There is no obvious reason for believing that the formation of a simple colon diverticulum will necessarily be the source of

symptoms. In fact, we frequently find evidence of diverticula on the *x*-ray plate without the patient presenting any local symptom. It is apparent that only when pathologic changes take place that are secondary to the diverticula do the patients

Fig. 255.—Portion of descending colon. Specimen laid open to show the internal orifices of the diverticula. (Taken from Maxwell Telling and Gruner, "British Journal of Surg.," vol. iv, No. 15.)

complain. It is difficult to estimate what percentage of diverticula have inflammatory changes such as to produce symptoms. Telling estimates that 60 per cent. of all persons having colon diverticula may have symptoms.

It is clear, therefore, that the recognition of inflammatory sequelæ is of utmost importance in the consideration of multiple diverticulitis. In general, the various secondary changes and complications are the result of infection through the walls of the diverticula. They have been classified for convenience in various ways. The following is as typical as any:

(*a*) *General peritonitis* may be produced by the actual passage into the peritoneal cavity of bacteria or their toxins through the thinned-out walls of the diverticula, some of the walls being so thin as to consist only of peritoneum.

(*b*) Acute gangrenous inflammation may result from strangulation at the neck of the pedunculated type of diverticulum.

(*c*) The chronic proliferative extramucosal inflammation is the most constant pathologic process. Telling considers this condition the most important. These proliferative changes produce in time large palpable masses. These masses tend to surround the gut. Embedded in them are the diverticula. Later they tend to actual intestinal obstruction. It is this type of inflammation to which the term "peridiverticulitis" applies, and it is this complication which is frequently diagnosed as cancer or sarcoma.

(*d*) It is evident that, with a mass of connective tissue in close proximity to other organs, adhesions involving these organs will arise. The bladder, small intestine, and pelvic organs are the viscera most commonly affected.

(*e*) *Perforation* is one of the most serious complications. This may be *acute*, perforation taking place through the thinned-out diverticulum wall or possibly through an ulcerated area. This type of perforation may follow severe strain. Chronic perforation may slowly take place into the peridiverticular mass of connective tissue. This may be associated with abscess formation. Fistulæ connecting the colon with the bladder or small intestine may follow a chronic perforation.

(*f*) Chronic inflammation of the mesentery as a complication of diverticulitis has been reported by several, among them Telling and McGrath. The late changes in mesenteritis are of importance, in that contracting bands of connective tissue in

the mesentery may be an etiologic factor in the production of some cases of volvulus.

(*g*) The development of *cancer* on the inflammatory mass is a possibility. The condition is analogous to the development of cancer on a chronic indurated gastric ulcer. McGrath reports that in his series of advanced peridiverticulitis with formation of a mass of connective tissue, 25.9 per cent. showed evidence of malignancy.

Beer, Wilson, Telling, Giffin, McGrath, Hartwell, and Cecil have all presented theories as to the cause of diverticula. The following factors are worth consideration:

1. Age: Most cases occur in people beyond middle life. Occasional cases are reported in patients under thirty. Ashhurst reports a possible case of seven years.

2. Males are twice as susceptible to diverticula as females.

3. Obesity seems to be a definite predisposing cause; on the other hand, rapid loss of weight may be followed by diverticulitis.

4. The normal muscle structure of the colon, particularly the arrangement of the muscle-fibers, is thought to predispose toward diverticulum formation.

5. The physiologic rôle of the sigmoid, in that it contains gas and fecal material under more or less pressure, may be an etiologic factor.

6. The points of entry for blood-vessels passing through the intestinal wall seem common sites for diverticula. These points of entry are also close to the appendices epiploicæ.

7. Another predisposing factor may be a variation in the size of the blood-vessels as found in congestion from venous obstruction, or high arterial pressure in cardiorenals or advanced arteriosclerosis.

8. There may be a congenital predisposition.

9. Simple muscle weakness in the intestinal wall may have a causal relation to the presence of diverticula.

One or all of the above factors may be involved in the production of diverticula. In general, herniæ of any kind are the result of *increased pressure* within a cavity plus a *local weakness* in the wall. In the region where diverticula most commonly

occur (the sigmoid) the intestine is subject to *increased pressure* from accumulating feces and gas.

Weakness in the wall may be produced by localized fatty degeneration of the muscle; degeneration from cachexia; the foramen for the blood-vessels with the coat of connective tissue are less resistant than the surrounding muscle-fiber, etc.

Multiple diverticulitis presents a fairly definite clinical picture. The average age of the patient is from forty-five to sixty and, in general, there is a marked tendency to obesity. In our cases diagnosed by the *x*-ray the majority of the patients weighed about 200 pounds. Men seemed more susceptible than women.

A history of chronic constipation is found in about one-quarter of the cases. A picture of chronic obstruction with alternating diarrhea and constipation is suggestive, the chronic obstruction being from the secondary connective-tissue changes.

Pain is present in a large percentage of cases. In the chronic type there is frequently a long history of attacks of crampy pain in the left lower quadrant. About 15 per cent. give a story of abdominal pain of considerable severity. It must be remembered that symptoms are the result of the secondary pathologic changes. These may vary all the way from acute peritonitis to acute obstruction, so that the symptoms of pain may be variously manifested.

The simple diverticula without secondary inflammation changes probably do not give symptoms.

A palpable tumor in the left side is found in about 30 per cent. of the cases giving symptoms. It is usually an elongated "sausage-shaped" mass lying commonly just above and more or less parallel with Poupart's ligament. Frequently it is mobile. A characteristic of this tumor is its variability. It may appear and disappear several times, its appearance being accompanied by signs of inflammation.

Abscess formation, local peritonitis, obstruction, etc., all show their usual clinical signs of fever, leukocytosis, pain, etc.

Rectal examination is negative in all cases except where there is actual diverticulum formation in the rectum itself. The proctoscope reveals nothing.

Multiple diverticulitis is mainly an extramucosal disease of the intestine, so that commonly the mucous membrane remains intact. As a result the passage of macroscopic blood is not commonly associated with diverticulitis. This is an important point in the differentiation of cancer. Carcinoma is a mucosal disease and associated with bleeding.

In the differential diagnosis carcinoma of the colon is the important condition to be ruled out. The pathologists tell us that possibly 25 per cent. of the chronic indurated diverticulitis cases become malignant.

But, in general, a patient who is well nourished and continues to maintain good nutrition in spite of symptoms; who has a long history of attacks of abdominal pain, localizing in the left lower quadrant; who presents at times a palpable mass in the left lower quadrant; usually who has not passed macroscopic blood; who may present evidence of vesical fistulæ; and with a negative sigmoidoscopic examination—such a patient may reasonably have made a diagnosis of chronic proliferative diverticulitis.

In carcinoma of the colon there is usually an early loss of flesh. Pain is not a constant symptom until obstruction sets in. Tenderness is a late finding. Blood, on the other hand, may be an early finding. When tumor is present, it is permanent.

From the other forms of pelvic inflammation it may be difficult to differentiate multiple diverticulitis. Left-sided appendix can usually be ruled out by the *x*-ray. In the presence of a palpable left-sided tumor one must always consider diverticulitis.

Up to the present time roentgenologists have perfected no special technic for visualizing colon diverticula. Such cases of multiple diverticulitis as have come to our attention have been found in the course of routine *x*-ray intestinal examination.

For those roentgenologists who do not make a routine practice of radiographing the abdomen previous to the adminstration of the barium meal we wish to emphasize the importance of this preliminary study. Just as preliminary routine plates of

the right upper quadrant will occasionally visualize a gall-stone, so plates of the left lower quadrant oftentimes will give evidence of importance in the question of multiple diverticulitis.

From the pathology we learn that fecaliths may be found in the diverticula due to the prolonged retention of fecal material. On account of the deposition of calcium the fecaliths are capable of casting distinct shadows on the *x*-ray plate. Unless preliminary plates are made, such shadows will be obscured by the barium meal.

Diverticula, which have become separated from the intestine, and are free bodies in the abdominal cavity, may produce a shadow on the plate. Their free movement will be demonstrated.

These preliminary plates may also reveal shadows, which later in the examination might lead to confusion, unless their presence had been previously recognized. Calculi in the left kidney or ureter; calcified glands, retroperitoneal or mesenteric; phleboliths; sclerosis of iliac arteries—all may produce shadows somewhat simulating the contents of diverticula.

The plates should be made both on the front and back. In our experience an intensifying screen allows us to use a little less penetrating ray, resulting in a more contrasty plate. This additional contrast, slight as it may be, frequently makes all the difference between showing and missing an obscure shadow. The fluoroscope helps in differentiating a movable and fixed shadow, and particularly in determining if it moves with respiration.

After the preliminary examination the patient is given the opaque meal. The meal which has been used routinely for several years in general gastro-intestinal examinations has proved satisfactory for the demonstration of colon diverticula. It consists of 2 glasses of buttermilk with $1\frac{1}{2}$ ounces of barium sulphate in each glass. This is taken on an empty stomach.

With the buttermilk meal the appendix is more readily filled than with other meals, so it is likely that diverticula are more readily filled with this type of meal.

Twenty-four hours after the meal we find the best filling of the colon. It is on this plate that the diverticula are first

visualized. As a frequent site for the diverticula is near the mesenteric attachment, it is obvious that in a simple antero-posterior plate these shadows may be hidden by the mass of barium filling the lumen of the colon. Palpation under the fluoroscopic screen or stereoscopic plates may bring to light some of these hidden diverticula (Figs. 256–259).

As these pockets tend to retain the barium over a prolonged period, we have found that plates made thirty-six to forty-eight

Fig. 256.—Normal colon. Plate made following a barium enema.

hours after the meal show the diverticula to the best advantage. At this time the lumen of the colon will be entirely free of the barium meal, and the barium-filled diverticula stand out clear and distinct.

It is of importance to determine the length of time barium is retained in these pockets. This, of course, is variable and de-

pends upon the type of diverticulum. We have observed cases where plates made not only days but weeks after the meal still showed evidence of barium in the diverticulum.

The examination with the barium meal should always be followed up with the barium enema. The enema is not as satisfactory as the meal for visualizing the actual diverticula. The pockets do not fill readily, perhaps for the same reason that the

Fig. 257.—Normal colon. Plate made twenty-four hours after a barium meal.

appendix is not filled by the enema. Then again the colon is usually so distended by the enema that the mass of barium very easily obscures many of the diverticula. The enema is of value, however, in bringing out some of the secondary pathologic changes, such as chronic thickening of the intestinal wall, with narrowing or beginning obstruction; abscess cavities, chronic perforation, fistulæ, etc.

Our practice is to mix up 5 or 6 ounces of barium with a pint

of buttermilk and water enough to make a quart. This is given slowly with very little pressure; the elevation of the container usually not more than 2 feet above the table. A soft rubber rectal tube is used, the end being inserted just beyond the internal sphincter. The patient, if possible, lies on the left side.

This solution is not irritating, and the patient usually retains it comfortably for ten minutes or more. Plates and study with

Fig. 258.—Normal colon. Plate made after a barium meal. Some hypertonicity. Note appearance of haustra in transverse colon, likely to be confused with diverticula.

the fluoroscope are made immediately. In fact, it occasionally gives valuable information to watch the abdomen with the fluoroscope as the enema is being given.

The patient is next asked to empty the colon as completely as possible, and further plates are made. These last plates will visualize any diverticula which retain the barium, and also

give us information as to the ability of the patient to empty the colon.

The various radiographic appearances of multiple diverticula roughly group themselves into three general classes. This radiographic classification corresponds with the grouping based on their pathology.

The *first class* is composed of cases showing simple diverticu-

Fig. 259.—Normal colon. Following a barium enema. Note the atonic condition of the intestinal walls.

lum formation. The diverticula may be diffusely scattered throughout the colon. The patient may or may not have symptoms.

The *second group* consists of those cases showing beginning secondary inflammatory changes. These cases usually show the diverticula pretty well localized about the descending colon and sigmoid. The patients usually are having symptoms.

The *third group* consists of cases showing advanced secondary inflammatory changes, such as pericolitis, tumor, obstruction, etc. (Figs. 273–277).

Group 1.—Occasionally, in the course of the routine gastrointestinal examination, one will observe in the plate made twenty-four hours after the meal round discrete shadows of the

Fig. 260.—Multiple diverticula. Plate made twenty-four hours after meal. Diffuse distribution of the diverticula over transverse colon, descending colon, and sigmoid. Simple diverticulum formation without inflammatory changes. Class I.

same density as the barium-filled colon, but usually distinct from it. The shadows vary in size from that of a pinhead to a dime or larger. They are circular in outline and are in close proximity to the intestinal wall. Under the fluoroscopic screen they seem to be a part of or intimately connected with the wall, yet distinctly outside the lumen of the intestine.

These shadows represent the barium-filled diverticula. They may be seen anywhere throughout the entire colon from the cecum to the rectum. Any portion of the circumference of the gut may be affected. As these diverticula have a tendency to retain the barium, as mentioned before, the best time to observe them is thirty-six to forty-eight hours after the meal, at which

Fig. 261.—Multiple diverticula. Plate made twenty-four hours after barium meal. A few simple diverticula diffusely distributed at *A*, *B*, and *D*. Appendix visualized at *C*. Patient has no symptoms. Class I.

time the lumen of the intestine is free of the barium and the round discrete shadows of the barium in the diverticula stand out clearly.

These shadows, varying in number, may be seen anywhere along the course of the colon, the most likely site being the descending colon and sigmoid. In some of the extreme cases the diverticula appear very much like a string of beads.

The diverticula classified in this first group are frequently not associated with symptoms. They are occasionally found during a routine examination in people apparently well.

As the condition is one of a simple diverticulum formation without inflammatory changes, about the only symptoms to be expected would be those of toxic absorption, the absorption being from the prolonged stasis of fecal material in the diverticula.

Fig. 262.—Multiple diverticula. Plate made twenty-four hours after barium meal. No symptoms; no inflammatory changes. Class I.

Two conditions may present radiographic pictures likely to be confused with simple diverticula. *First*, a hypertonic condition of the colon may be associated with almost a spastic condition of the haustra, so that discrete masses of barium become separated from the mass of barium in the lumen. The dependent portions of the haustra of the transverse colon seem to be a common location. The plates made at intervals will demonstrate

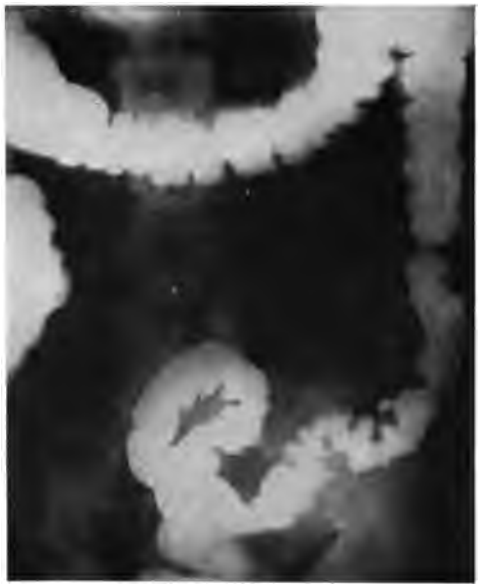

Fig. 263.—Simple diverticulum. Plate taken after barium enema. Class I.

Fig. 264.—Multiple diverticula. Plate made after a barium enema. Two diverticula seen. Class I.

that these apparent sacculations are only temporary. A true diverticulum is permanent. Again, study with the fluoroscope and barium enema will show that these haustral shadows are within the lumen of the gut and are not extramural, as is the case with the diverticulum.

Second, in a certain type of individuals the colon contents become subdivided into small discrete masses, particularly in

Fig. 265.—Multiple diverticula after a barium enema. A few small diverticula. Class I.

the descending colon and sigmoid. A spastic type of constipation frequently produces this condition. These small masses may be confused with diverticula. In inconstancy of these shadows is the chief means of differentiation.

Group 2.—The second group in the classification of diverticulitis is composed of cases showing beginning secondary changes of an inflammatory nature. The diverticula in this

group are usually localized along the lower descending colon and sigmoid. The patients invariably have more or less definite symptoms.

The *x*-ray plate shows numerous diverticula ranging from 4 or 5 to 20, situated in a limited region about the junction of the sigmoid and descending colon (Figs. 266–272).

The amount of intestine involved is not more than 2 or 3 inches. The important radiographic appearance differen-

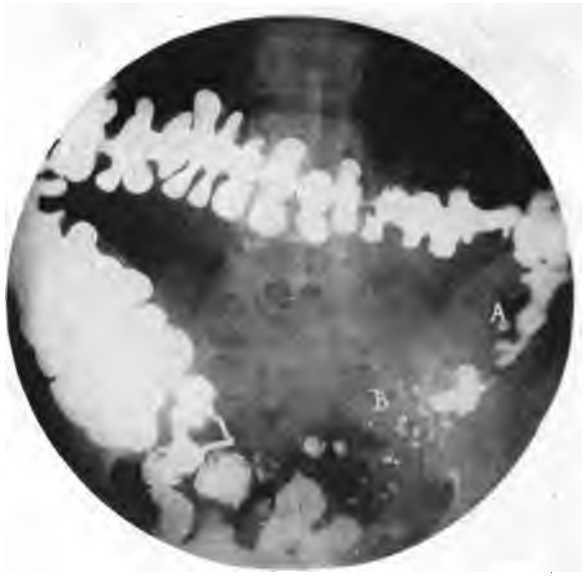

Fig. 266.—Multiple diverticula. Plate made twenty-four hours after barium meal. Note that the diverticula are definitely localized at junction of descending colon and sigmoid *A* and *B*. There is narrowing of the lumen of the colon from inflammatory tissue and spasm. Class II.

tiating this from the preceding group is a beginning narrowing of the lumen of the intestine. The apparent narrowing may at first be wholly due to spasm. It is usually best observed following the barium meal. Frequently the enema fails to reveal this spastic condition. In later cases, where there is actual thickening about the colon wall, the narrowing is constant both with the meal and enema.

Fig. 267.—Multiple diverticula. Condition similar to Fig. 266. Class II.

Fig. 268.—Multiple diverticula with beginning narrowing of the colon at *A*. Plate made following the barium enema. Rather diffuse distribution of the diverticula throughout the descending colon and sigmoid. Class II.

Under the fluoroscopic screen there is usually found localized tenderness. The sigmoid may be fixed from inflammatory adhesions. The infiltration of chronic inflammatory tissue in and about the intestinal wall will produce a demonstrable rigidity of the wall.

The condition which it is important to differentiate from cases

Fig. 269.—Multiple diverticula. Plate made after barium enema. Localized diverticula at *A*, with narrowing of the lumen mainly due to spasm. Phleboliths at *B* might be confused with diverticula, unless their presence had been previously recognized in the "preliminary examination."

in this group and the third group is *carcinoma*. I do not believe that a positive differentiation can always be made with the x-ray alone, particularly as a certain number of cases have malignant disease developing on top of a chronic diverticulitis. The history and physical examination as well as laboratory findings are of the utmost importance in making this differentia-

tion. In studying the *x*-ray plates there are one or two points which one must bear in mind when considering the possibility of cancer or diverticulitis.

In the first place, the *x*-ray appearance of diverticulitis tends to remain constant even over long periods of time. If malignant disease is present, repeated examinations show a progress-

Fig. 270.—Multiple diverticula. Plate made after the barium meal. Isolated diverticula at *A* and *B*. Secondary inflammatory changes involving descending colon. Class II.

ing lesion. If in a continued study of a diverticulitis case we find a portion of the involved area beginning to change its contour, one must be suspicious of a beginning malignancy.

Cancer, in general, is an intra-intestinal growth. The growth is in the wall of the colon, extending into the lumen and definitely circumscribed. The tumor mass in diverticulitis is out-

side the intestinal wall, being a pericolitis or perisigmoiditis. It is not definitely circumscribed.

Hence when we find a filling defect in the colon producing a narrowing of the lumen, with the transition from normal bowel to diseased bowel, immediately we suspect cancer. With a narrowing due to diverticulitis several inches of colon on either side of the actual point of narrowing will show more or less

Fig. 271.—Localized diverticula formation. Plate made after barium enema. Two diverticula at *A* and *B*. Narrowing of the colon at this point. Symptoms of chronic obstruction. Class II.

evidence of disease. In other words, in diverticulitis the transition from normal bowel to diseased bowel is *gradual*, while in cancer the transition is abrupt (Figs. 273 and 279).

Obstruction from cancer is likely to be severe, rapidly progressive, and finally complete. Obstruction from chronic diverticulitis is usually not severe, with a very gradual progress extending sometimes over a period of years, and rarely complete (Figs. 278 and 280).

Under the fluoroscopic screen an intermittent palpable tumor of the colon is always peridiverticulitis. A constant and persistant tumor mass may be cancer.

Group 3.—In the third group we put the cases showing advanced secondary changes associated with moderate to severe symptoms. In this group are the various forms of obstruction.

Fig. 272.—Diverticula at *A* and *B*, with narrowing of the colon at *B*. Barium enema. Class II.

The tendency in any chronic inflammatory mass is to contract. As we find a chronic obstruction following an indurated ulcer at the pylorus of the stomach, so with the inflammatory condition about the colon the same process takes place. The tendency in chronic diverticulitis is toward chronic obstruction, possibly extending over years.

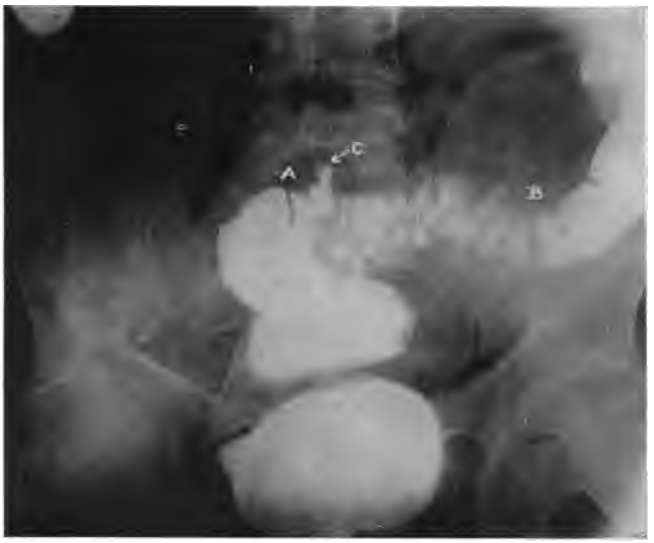

Fig. 273.—Diverticula with advanced inflammatory changes. Plates taken following the barium enema. Infiltration of intestinal wall from *A* to *B*. One large diverticulum at *C*. Symptoms of obstruction. Operation showed a chronic inflammatory mass. Class III.

Fig. 274.—Diverticula with advanced inflammatory changes. Condition similar to Fig. 273. Class III.

Fig. 275.—Multiple diverticula with chronic inflammatory changes. Plate taken after a barium enema. Narrowing of intestine from *A* to *B*. Operation showed tumor mass composed of chronic inflammatory tissue. Class III.

Fig. 276.—Chronic inflammatory changes involving descending colon and sigmoid. No diverticula are seen, but the intestine presents a serrated appearance such as is found associated with diverticulitis. Class III.

Fig. 277.—Chronic inflammatory changes about the descending colon and sigmoid. Plate taken after a barium enema. The "serrated" appearance is more marked than in the previous illustration. The individual diverticula are not seen. Class III.

Fig. 278.—Same case as Fig. 277, showing constriction at point *A*. Operation showed this to be chronic inflammatory tissue.

The *x*-ray picture of colon obstruction is characteristic and needs no special comment. There is stasis proximal to the point of obstruction with possible dilatation of this portion of the intestine. This is studied best following the barium meal. Plates should be taken at sufficient intervals to determine the

Fig. 279.—Cancer of the colon at point *A*; *B*, transverse colon; *C*, hepatic flexure; *D*, appendix. Plate taken after barium enema. Note the abrupt transition from normal bowel to diseased bowel, which is characteristic of growth. In Fig. 278 note that the transition from normal to diseased bowel is gradual. This is characteristic of chronic inflammation.

degree of obstruction—forty-eight or seventy-two hours after the meal, if necessary. The barium enema will confirm the meal and also give more detailed evidence as to the amount of colon involved and the degree of narrowing.

In this connection one must mention an appearance of the

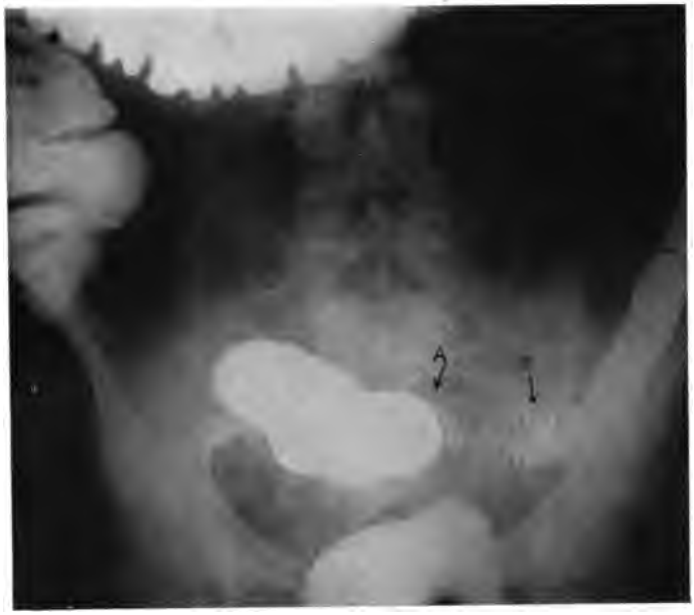

Fig. 280.—Chronic obstruction from diverticulitis. Lesion at points *A* and *B*.

Fig. 281.—Same case as Fig. 280 four years later, showing complete obstruction to barium enema. Operation revealed chronic inflammatory mass.

colon which has become to our mind almost pathognomonic of chronic diverticulitis, although the diverticula themselves are not visualized. Particularly along the descending colon and sigmoid one occasionally finds a peculiar serrated appearance of the colon (Fig. 276). This may extend over several inches of the gut, and is associated with more or less narrowing of the lumen. These serrations are small, close together, and with a rather sharp point, presenting at times a saw-tooth appearance.

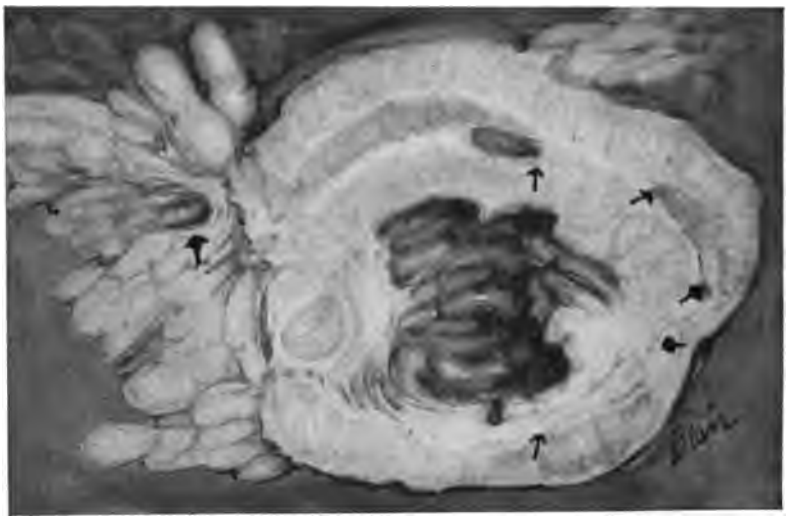

Fig. 282.—Drawing of specimen taken from preceding case. Shows cross-section of sigmoid, the wall of which is enormously thickened by chronic inflammatory tissue. Within the mass are seen the diverticula, to which the arrows are pointing. No evidence of malignancy.

They will not be confused with haustral shadows, for they are too numerous and too small, and, more important, they are constant in shape and position.

This serrated appearance is best seen following the enema, although it may be observed in the twenty-four-hour plate after the meal. Repeated enemata given on different days show this appearance to be constant.

The serrations do not change in size or shape. Further-

more, palpation under the fluoroscopic screen usually shows us that the intestine is more or less like a rigid tube. The walls are thickened and have lost their normal flexibility.

One infers that the serrated appearance is due to inflammatory thickening and induration in and about the intestinal wall, secondary to the presence of diverticula. The diverticula themselves not being visible, perhaps obliterated by the connective-tissue formation, or already so filled with mucus and fecal material that the barium is unable to enter.

There are other complications secondary to diverticulitis besides simple obstruction. Adhesions arising from the localized diverticulitis may involve other coils of intestines or other organs. Such a condition is best observed under the fluoroscopic screen.

Chronic perforation with abscess formation is not an uncommon sequel. The x-ray appearance is no different from a chronic perforation of gastric or duodenal ulcer. The plate may show an abscess cavity filled with barium outside the lumen of the gut, with a small isthmus of barium connecting it with the intestine. Acute perforations do not come to the x-ray man.

Some of the acute diverticulitis attacks simulate a left-sided appendix. The barium enema may be of help in such cases by demonstrating that the cecum and appendix are in normal position on the right side.

Rarely fistulæ may result from diverticulitis. Several cases have been reported of fistulæ connecting the bladder with the colon. Such conditions would be clearly shown on the x-ray plate.

In general, it must be borne in mind that the diagnosis of multiple diverticulitis is not to be made from the x-ray alone, although in some cases it may seem possible. The x-ray evidence must be considered in conjunction with the history, physical examination, and other laboratory findings. This is of particular importance in the differentiation of cancer and multiple diverticulitis.

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HARVARD MEDICAL SCHOOL

SOME FALLACIES IN THE DIAGNOSIS OF "PTOMAIN POISONING"

THE term "ptomain poisoning" has become both popular and vague. Jordan aptly states that it is a convenient refuge from etiologic uncertainty. In Europe the diagnosis of "ptomain poisoning" is seldom made, and the use of the term in any connection is very rare and quite guarded. In this country, however, cases of "ptomain poisoning" are still described in the medical journals, but more frequently in the lay press. Any acute gastro-intestinal attack resulting from a great variety of causes is apt to be called "ptomain poisoning." The diagnosis has fallen into disrepute because it lacks precision and has become a cloak for ignorance.

Selmi, in 1873, first used the word ptomain (from *ptoma*, a corpse) to include the poisonous products of putrefaction which gave the reaction then looked upon as characteristic of vegetable alkaloids. From the time of Selmi, when ptomains were regarded as animal alkaloids, our conception of these substances has changed markedly. The last attempt to give precision to the term was by Vaughan, who defined ptomains as intermediate cleavage products of protein decomposition.

For the past two years I have had charge of an investigation on the subject of "ptomain poisoning," in which I have been ably assisted by my associates in the Department of Preventive Medicine and Hygiene at the Harvard Medical School, especially by Professor J. Bronfenbrenner, Dr. D. L. Sisco, Dr. R. M. Atwater, Dr. John Weinzirl, Dr. E. W. Cheyney, Dr. W. G. Webber, Mr. M. J. Schlesinger, Mr. P. F. Orr, Miss C. R.

Davis, Miss H. Husted, Miss E. Wolff, and a group of others working upon the chemistry, bacteriology, and epidemiology of the subject of food poisoning. During the course of this investigation a special effort was made to discover and study every case occurring in and around Boston. As a result of these studies a number of interesting facts were discovered, which will form the basis of an extensive report. Here I want to call attention to some curious fallacies in the diagnosis of "ptomain poisoning." A careful study disclosed the fact that some of the cases were bacterial infections, others were caused by corrosive or irritating chemical substances, and still others were much wider from the mark.

We have searched in vain for "ptomains" that might cause gastro-intestinal or other symptoms. Split products of protein putrefaction are readily isolated. Some of these products have physiologic activity, but none of them so far have been demonstrated to be poisonous when taken by the mouth. The so-called ptomains isolated and described by Selmi, Nencki, Breiger, Schmiederberg, Faust, and Vaughan were usually obtained from putrid organic matter that had decomposed past the point where it would be used as food. Furthermore, most of these substances were tested by injecting them subcutaneously or intravenously into animals. Many substances are poisonous when thus introduced parenterally, although they may be harmless by the mouth. Again, many of the so-called ptomains isolated and described have since been shown to contain impurities. Chemists are now seldom confident of the purity of protein fractions, even when obtained in crystalline form. The chemical search for split protein products as the cause of "ptomain poisoning" has practically been abandoned. Most of these split products are amins, which are either not poisonous at all, or no more so than their corresponding ammonia salts. The chemical resemblance between muscarin and cholin has directed the work toward the phosphatids, but thus far this line of research has not helped solve the puzzle of "ptomain poisoning."

I have selected some instructive cases illustrating fallacies in the diagnosis of "ptomain poisoning" which have come to

our notice during the past two years in the investigations which we are carrying on at Harvard. Most of the following cases we have investigated personally; information concerning some of them was obtained by correspondence or from the literature.

Oxalic Acid Poisoning.—Our case No. 1, A. V. D., female, age fifty-eight. Ate about a half-peck of cooked rhubarb leaves (tops) Monday, May 14, 1917, at 4 P. M. Also took much of the water in which the leaves were cooked. Patient was sick all night; started to vomit about 3 A. M. on the morning of the 15th. At the same time diarrhea commenced and continued during the night, but bowels did not move again until the time of death. Patient vomited throughout the entire illness, was very thirsty, and drank a great deal of water. Temperature normal. Patient had had chronic pains in abdomen for many years, which were much intensified during this period. Died in ten days. Rigor mortis did not set in until thirty hours after death. Bacteriologic examination of material obtained post-mortem by rectal swab showed that the case was complicated with dysentery.

A brother-in-law of the patient ate some of the greens and was sick all night, but recovered. A sister of the patient ate a very small amount of the greens, and had but slight malaise.

Diagnosis: Oxalic acid poisoning. This case was investigated by us because it was reported as "ptomain poisoning."

A similar instance was reported in the Weekly Bulletin of the New York City Department of Health September 16, 1916. This was suspected "ptomain poisoning" due to sour-grass soup. The report follows:

"Some time ago, through our Weekly Bulletin, we asked physicians to be on the lookout for cases of ptomain poisoning, and to promptly report all such cases to the Department of Health. On investigation, very few of the cases proved to be really ptomain poisoning.

"A recent investigation showed that several cases of illness occurring in the East Side and originally thought to be due to ptomain poisoning were really due to sour-grass soup. The soup was prepared from 'Qchav' or 'Szchav' leaves, more commonly

known as sour-grass—a species of sorrel. This plant is rich in oxalic acid salts, analysis in the Health Department's chemical laboratory showing as much as 2 grains of oxalic acid in 1 ounce of the leaves, and double that in the stems.

"An inspector of the Health Department's Bureau of Food and Drugs found that sour-grass soup is a common dish in some of the East Side restaurants. In some cases the stalks of the leaves are discarded as too acid. In one restaurant the inspector found that the stalks were removed, the leaves then well washed, and the washings rejected. After that the leaves were soaked in cold water overnight and then boiled in water for fifteen minutes. This extract was poured off and rejected. A second boiling of the leaves then followed, after which eggs and cream were added. The dish was then ready to serve. Chemical analysis of the finished soup showed about *10 grains of oxalic acid per pint of soup.*"

Belonging to this group of irritating chemical substances is the following account which we obtained from Chicago:

Tartar Emetic.—During the summer of 1917 a number of cases of "ptomain poisoning" occurred in Chicago due to tartar emetic. This drug was given to guests in hotel dining-rooms and restaurants who had slighted waiters in giving "tips." Ten waiters were indicted by the Grand Jury. The Assistant States Attorney wrote me as follows: "That 'Micky Finn' powders were given to guests at banquets and in hotels and restaurants is well established by the evidence in possession of this office. The analysis shows the powder to be pure salt of antimony and potassium nitrate, known by the trade as tartar emetic."

Saltpeter.—Our case No. 5. This was an outbreak of diarrhea occurring during June and July, 1917, in the 6th Massachusetts Regiment, at Camp Darling, Framingham, Mass. We were asked to investigate a small outbreak of diarrhea at the camp, and I sent Dr. Atwater there on July 9, 1917. The symptoms were mild and without fever. A study was made of the blood and feces and also of the food, milk, and water. Nothing indicative was discovered chemically or bacteriologically. Suspicion was then aroused toward some mischief maker with

malicious intent. Finally, an old Navy surgeon suggested looking into the sugar. It was found to be somewhat unusual in taste. Dr. Webber found a sample of this sugar to contain about 1 per cent. of potassium nitrate. It is assumed that the saltpeter was added to the sugar as an anaphrodisiac. This ineffectual attempt at chemical morality cannot meet with the approval of the true reformer. The sugar was changed and the trouble ceased.

Uremia and Neoplasm.—Our case No. 56. This illustrates loosely and vaguely the term "ptomain poisoning" is used even in one of our best and oldest medical journals. The following editorial appeared in the Boston Medical and Surgical Journal of February 13, 1919, p. 204:

"Dr. Thomas F. Harrington, Deputy Commissioner of the State Board of Labor and Industries, died at his home in Boston, January 19, 1919, after a five weeks' illness from ptomain poisoning."

Investigations made by Dr. Sisco disclosed the fact that Dr. Harrington thought he was troubled with "ptomain poisoning," and this must have been the origin of the diagnosis. The true facts appeared in an editorial correction in the Boston Medical and Surgical Journal of April 3, 1919, p. 398:

"In a recent issue of this Journal was published an obituary of Dr. Thomas F. Harrington in which it was said that the cause of his death was "ptomain poisoning." Further investigation of this diagnosis, with the physician in attendance upon Dr. Harrington and the death certificate, has disclosed, first, that the origin of the diagnosis is unknown, and second, that the immediate cause of death was uremia, an apparent result of a neoplasm which obstructed the ureters."

The report of Dr. Harrington's last illness is a very good illustration to show how deep-rooted the fallacy of "ptomain poisoning" has grown, and how loosely the term is used.

In the Journal of the American Medical Association, March 17, 1917, one of the causes of chronic nephritis is given as "repeated ptomain poisoning."

Dysentery.—Our case No. 52. One of the early instances

of "ptomain poisoning" investigated by us was a family outbreak which turned out to be bacillary dysentery. The following is a condensed account of the essential features:

Wood family consisted of mother, father, and four children, all living in Avon, Mass. On August 23, 1918, the entire family went to visit the wife's parents at Randolph. On the 27th the baby was taken sick, having malaise, diarrhea, and fever as the chief symptoms. The child tossed its head about a great deal during its sickness. No blood was noticed in the stools. Child died on September 1st. No autopsy was done.

On the 26th of August Gladys (nine years old) had abdominal pains and a profuse diarrhea with movements every fifteen minutes. The stools were bloody and contained mucus. Later there was vomiting and fever. Child was sick until September 3d.

On September 1st Thirza (three years old) began to have a bloody diarrhea with fever. There was also a twitching and tossing of the head. She died September 9th, having vomited the morning death took place.

On September 3d Thelma (seven years old) developed bloody diarrhea, which did not last very long.

Mother and father each had a slight attack of diarrhea, but not of long duration or great intensity. Same was true of the grandparents.

Investigation of diet showed that meat and salt fish had been brought with the family from Avon, but that otherwise food eaten was purchased in Randolph. Family suspected overripe cantaloup, but no evidence was found to support this.

It was found that there had been a number of such cases of sickness in Randolph and several deaths.

An autopsy was performed on Thirza Wood, and an account of the findings disclosed lesions of bacillary dysentery. Bacteriologic examination of the intestines gave a positive culture of the dysentery bacillus (Shiga).

Bacterial Infections.—We now know that most of the cases formerly called "ptomain poisoning" are, in fact, instances of bacterial infections. It is not even proper to call such cases

food poisoning. No one would think of speaking of a case of typhoid fever contracted from milk as food poisoning. There is in this category a group of gastro-intestinal disturbances caused by the *Bacillus enteritidis* and its congeners. Gärtner, 1888, described an outbreak associated with the *Bacillus enteritidis* which has since been called the Gärtner bacillus. This micro-organism belongs to the colon-typhoid group, and is the type of a number of closely associated, Gram-negative, spore-free bacilli which have a certain amount of pathogenic power. They are taken into the system with food and drink; direct and indirect infection occurs; food, fingers, and flies play a rôle in the transfer of these infections, as they do in the case of typhoid fever. Bacillus carriers of this group are suspected, but are probably rare. Infections of this type bear a close resemblance to paratyphoid fever. They are much more common abroad than in this country; at least, they are reported much more often from England, Germany, and France. These outbreaks are very apt to be associated with church suppers, sorority picnics, and fraternity dinners. The reasons seem self-evident when we investigate the way the food is often prepared, handled, kept, and served at such functions. It is no longer the fashion to call bacterial infections belonging to this class "ptomain poisoning," for they have a distinct etiologic standing of their own. We are making an extended study of this phase of the subject, and will have a report upon it at a later time.

Indigestion.—Our case No. 8, M. J. R., male, forty-nine years, played golf on July 23, 1917, a particularly warm and humid day. While fatigued and overheated ate an ear of boiled green corn, ham omelet, cold slaw with mayonnaise, potatoes, watermelon, and cake. During the night had abdominal pain in region of the umbilicus. At daybreak took a Seidlitz powder, and this was followed by two or three loose semiformed movements, not noticed to contain blood or mucus. Felt weak and tired; no nausea, no vomiting, but a slight headache added to the effect of the malaise.

Temperature next morning, July 24th, 101° F. Entered hospital about noon. On admission, pulse 100, temperature

100.6° F. Was given calomel, six doses of 10 milligrams each, at fifteen-minute intervals. The temperature rose to 101.2° F. that evening, but the next morning was 99° F., and later the same day returned to 98.6° F. and remained normal. No further symptoms.

White cell count was 5200. Stools contained Bacillus coli and B. coli communior, but no pathogenic bacteria.

The diagnosis was doubtless a mild heat exhaustion, accentuated by acute indigestion due to indiscretion in diet, and, furthermore, eating too quickly while fatigued and overheated.

Acute digestive troubles with a great variety of symptoms and from a great variety of causes are often loosely and carelessly called "ptomain poisoning."

Nervous Diarrhea.—Our case No. 10. On March 15, 1918, Dr. X. ate some experimental food at a luncheon club, which is maintained at the Department of Preventive Medicine at the Harvard Medical School for the express purpose of studying the problems in question. Fifteen minutes later Dr. X. became very nervous and worried about his condition, questioning the other men in the laboratory concerning his appearance, etc., and watching himself closely for symptoms of illness. His apprehension about himself was accentuated by injudicious teasing on the part of his colleagues. Within about two hours he developed a moderate diarrhea which lasted for several hours. There were no other symptoms.

The experimental food was canned corn, and was eaten by three other individuals, none of whom complained of illness. The analysis of these cans by Dr. Weinzirl revealed no organisms. Dr. X.'s dietary for the preceding five days contained nothing which would be looked upon with suspicion.

His diarrhea was doubtless of "nervous" origin, especially as he gave a history of similar attacks brought on by emotion or excitement.

Botulism.—Recently three deaths occurred in Spokane, Washington, which were reported as due to "ptomain poisoning." Upon investigation they were shown to be botulism, said to be due to home-processed beets.

Botulism is a true toxemia. It is caused by a toxin which is produced by the Bacillus botulinus in nitrogenous food. The toxin is produced at room temperature in vegetables and fruits as well as in sausage and other meats. It has a striking symptomatology and is often fatal, but is a very rare cause of food poisoning. Botulinus poison is a true toxin, and corresponds in all essential respects to the toxin of diphtheria and the toxin of tetanus, the only difference being that it is poisonous when taken by the mouth, whereas the toxins of diphtheria and tetanus are only poisonous when injected into the tissues. It is just as irrational to call botulism "ptomain poinsoing" as it would be to call diphtheria or tetanus by this name.

Anaphylaxis.—Anaphylactic reactions to certain foods are of very common occurrence. These reactions are sometimes severe and are accompanied by urticarial eruptions of the mucous membrane as well as of the skin. These changes may take place in the esophagus and perhaps the stomach, for it is not uncommon to have vomiting in severe cases, and the vomitus sometimes contains blood. The attacks are usually severe and come on suddenly, and in a number of instances have been mistaken for "ptomain poisoning." Persons who are hypersusceptible to certain foods, such as shell-fish, strawberries, etc., sometimes associate these symptoms, and the literature contains a number of instances of this condition being mistaken for "ptomain poisoning."

Oxalic acid, tartar emetic, and saltpeter; uremia and cancer; dysentery and bacterial infections; indigestion, nervous diarrhea, botulism, and anaphylaxis have all been mistaken for "ptomain poisoning." It is not necessary to give further instances showing the fallacies in this diagnosis. The term should be avoided, for it lacks precision. In fact, "ptomain" is a term for a chemical substance of uncertain origin, unknown nature, and doubtful existence.

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(Published July, 1914)

A Text-Book of Medical Diagnosis. By JAMES M. ANDERS, M. D., PH. D., LL.D., Professor of Medicine, and L. NAPOLEON BOSTON, M.D., Professor of Physical Diagnosis, Medico-Chirurgical College, Graduate School of Medicine, University of Pennsylvania. Octavo of 1248 pages, with 466 illustrations. Cloth, $6.00 net.

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FOURTH EDITION

This new edition has been carefully revised, making it still more useful than the two editions previously exhausted. The articles on milk and alcohol have been rewritten, additions made to those on tuberculosis, the salt-free diet, and rectal feeding, and several tables added, including Winton's, showing the composition of diabetic foods.

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NEW (3d) EDITION—published April, 1917

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SECOND EDITION, RESET

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Cohen and Eshner's Diagnosis.

Second Revised Edition, 1900

ESSENTIALS OF DIAGNOSIS. By S. SOLIS-COHEN, M. D., Senior Assistant Professor in Clinical Medicine, Jefferson Medical College, Phila.; and A. A. ESHNER, M. D., Professor of Clinical Medicine, Philadelphia Polyclinic. Post-octavo, 382 pages; 55 illustrations. Cloth, $1.25 net. *In Saunders' Question-Compend Series.*

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Kelly's Cyclopedia of American Medical Biography

CYCLOPEDIA OF AMERICAN MEDICAL BIOGRAPHY. By HOWARD A. KELLY, M. D., Johns Hopkins University. Two octavos of 525 pages each, with portraits. Per set: Cloth, $10.00 net; Half Morocco, $13.00 net. **Published April, 1912**

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Illustrated

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ESSENTIALS OF PRESCRIPTION WRITING. By CARY EGGLESTON, M. D., Instructor in Pharmacology, Cornell University Medical College. 16mo of 134 pages. Cloth, $1.25 net. **Second Edition published November, 1917**

Slade's Physical Examination and Diagnostic Anatomy

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